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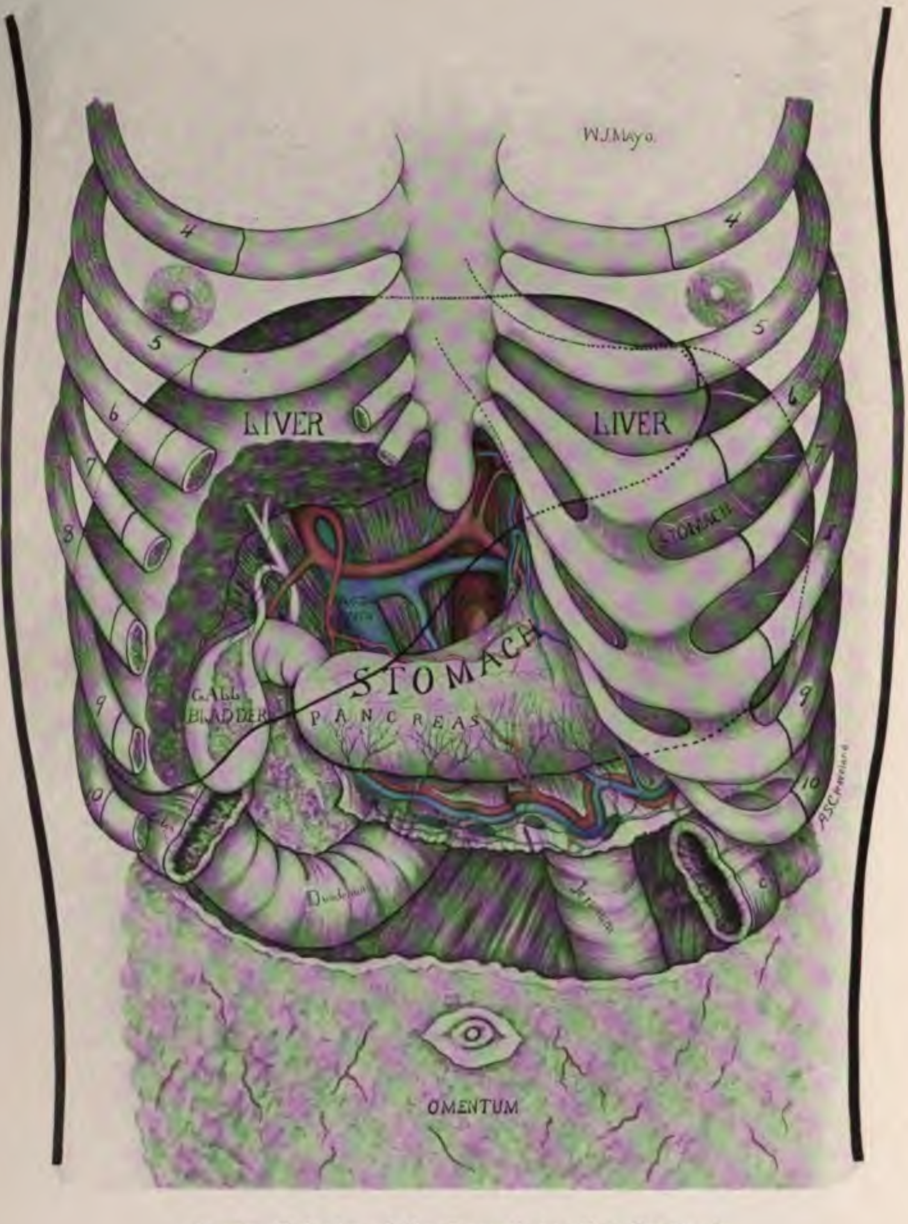

Showing Relation of the Stomach to other Organs in Abdominal Cavity.

(By courtesy of Dr. William J. Mayo.)

DISEASES OF THE STOMACH

BY

DR. I. BOAS

SPECIALIST IN GASTRO-ENTERIC DISEASES IN BERLIN

THE SOLE AUTHORIZED ENGLISH-AMERICAN EDITION
FROM THE LATEST GERMAN EDITION

BY

ALBERT BERNHEIM, M.D. (FREIBURG, GERMANY.)

PHILADELPHIA

Assistant to the late Dr. D. D. Stewart at the Polyclinic Hospital and Post-Graduate School, as Instructor in the Department of Diseases of the Stomach and Intestines; Privileged Physician to the Jewish Hospital of Philadelphia; Examining Physician to the Free Hospital for Poor Consumptives, Philadelphia-White Haven; Consulting Physician to the Nazarene Home for the Aged; ex-President of The Northern Medical Association of Philadelphia.

Illustrated with Five Full-page Plates and Sixty-five Engravings in the Text

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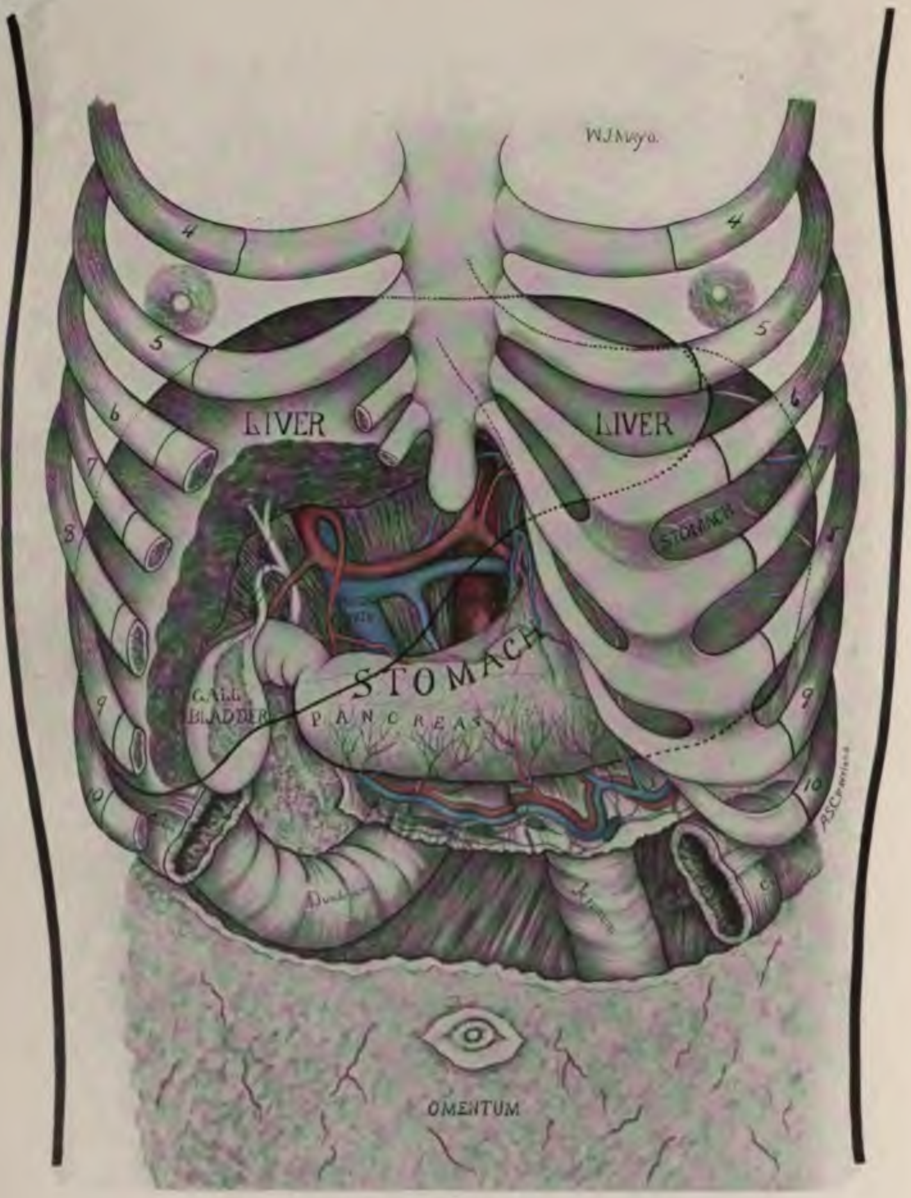

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AUTHOR'S PREFACE

TO THE ENGLISH-AMERICAN EDITION

AFTER my text-book on "Diseases of the Intestines" had been translated into English several years ago, I consider it a most particular satisfaction that now the same honour is conferred upon my work on "Diagnostics and Therapeutics of the Diseases of the Stomach."

Both books are the result of my own views, my own experience and partly of my own research work. In these factors lies their strength and, as you may say, their weakness. But in the dilemma of conscientiously registering all scientific acquisitions in a certain sphere and gathering them together in an iridescent bouquet, or of sifting and collecting that which, according to my own experience, has proved useful, there was for me never the least doubt in the selection.

There is still another feature which if I am not mistaken has aided my text-books in their success in Germany as well as in foreign countries: namely, the form of presentation, which is free from obscure hypotheses, easily comprehensible, in one word, popular.

In this form of presentation I see a regardful consideration for the reader, a factor that does not need any particular explanation on my side. A book may contain errors, and even mistakes—and what book is entirely free from them—but the reader must be enabled to recognize clearly the views of the author from the contents and form. Oracular phraseology which is understood only after repeated reading, and sometimes even not then, unprecise attitudes or a cautious neutrality always shows that the writer does not master his subject or at least does not possess the courage to endow his own conviction with a pregnant expression.

Although hereby undoubtedly an occasionally exaggerated subjectivism takes place in his views, this is rather an advantage than a disadvantage from the standpoint of the student. There develops between writer and reader some kind of intellectual *rapport* which at least to a certain degree may supplement and replace the lack of oral teaching.

I venture to hope that these features, dominant in the presentation of this work, will also meet with the approval and approbation of my American and English friends and colleagues.

I desire to extend my thanks to Dr. Bernheim for having undertaken the work of translation and carried it to a successful conclusion in this English-American Edition.

I. BOAS.

Berlin, Germany, April, 1907.

54308

TRANSLATOR'S PREFACE

Dr. Boas's Treatise on Diseases of the Stomach has experienced a most astonishing popularity, and that not only in Germany but also in other European countries. It has been translated into Russian, Italian, and Spanish, a fact which by itself is praise enough for the book; I trust that the book in its English dress will find the same acknowledgment as it has found abroad.

It is to my greatest regret that I cannot carry out the idea which was presented to me when this translation was started. At that time my friend and teacher, Dr. D. D. Stewart, promised me to write the foreword to Dr. Boas's Diseases of the Stomach. But Dr. Stewart died before he could fulfil his promise. Here, I wish to take the opportunity to give expression of thanks to and of highest appreciation of the late D. D. Stewart, in whose death the medical profession in general and the specialists in gastro-enteric affections in particular have suffered a great loss. Dr. Stewart was one of the pioneers in this work in this country. His memory will always be kept sacred, wherever the study of the Diseases of the Stomach is a matter of thought.

Dr. Boas's book is, as Dr. Boas himself in his preface says, particularly a book for the general practitioner, but also for the specialist and for the student. Many physicians who took a post-graduate course in the Philadelphia Polyclinic, have often expressed a desire for a translation of Boas's book.

In the last few years much work pertaining to gastro-enteric diseases has been done in this country; but to have incorporated all the publications of American authors would have made the book too large, and therefore the translator has made but few additions, which are indicated by brackets [ ], and especially has taken notice of the progress made by American surgeons.

It gives me great pleasure to express my cordial thanks to Dr. William J. Mayo and Dr. George E. Pfahler for their kindness in allowing me the use of the beautiful full-page plate illustration and of the excellent skiagraphs for this book, and to Drs. Walther E. Rahte and Milton K. Meyers for their assistance in the preparation of the translation, and to F. A. Davis Company for many favours extended to me by them.

Albert Bernheim.

1411 Spruce Street,
Philadelphia, May, 1907.

PREFACE

TO THE FIFTH GERMAN EDITION

THE large propagation which this present text-book found among the medical profession in Germany and foreign countries, and the increasing number of editions make it my special duty to pay increased care and attention to its improvement and revision. But to be sure even the specialist—since the number of publications have become hardly surveyable—has found the task becoming continuously more difficult, to do justice to the advances of science on the one side and not to lose oneself in specialistic subtleties on the other side. Neither must it be forgotten that this book does not intend to give a complete presentation of the pathology of the stomach, but merely intends to consider those methods and results of investigation which are in direct and immediate connection with the diagnosis and treatment of the diseases of the stomach. This standpoint may be my excuse to those investigators whose publications have not received any attention or not enough attention.

Otherwise in the preparation of this new edition I was guided by the same principles as in the former editions—namely: Hypotheses as few as possible, but practical hints in precise, easily comprehensive form as many as possible.

I hope that nothing of the subjective stamp which I endeavoured to give to the previous editions, has been lost in the new one. Only hereby the mental *rapport* between author and reader may be made a fact, a *rapport* which stamps a book of teaching as a book of learning.

I wish to express my sincere thanks to my assistant, Dr. Hans Ury, for the manifold aid given me in the preparing of this edition as well as for the preparing of the list of authors and subjects.

THE AUTHOR.

Berlin, May, 1903.

PREFACE

TO THE FIRST GERMAN EDITION

THE present treatise owes its origin to the fact that so many of the attendants in my practical courses repeatedly expressed their desire for a short and comprehensive presentation of the modern diagnostics and therapeutics of the diseases of the stomach.

After a longer hesitation I concluded to publish the same, particularly because I was induced to do so by the very agreeable perception that also many older physicians not familiar with the more recent methods of examination and treatment showed a continued and earnest interest towards this special branch.

This treatise in form and contents is especially adapted for the general practitioner, to whom it conveys the acquisitions of the modern diagnostic and therapeutic methods in conciseness and yet completeness, acquisitions which may be designated with pride as the fruit of German labour and German diligence.

As physician familiar with the needs of daily practice, and directed by my colleagues to those facts which have mostly escaped those kept away from the clinical auditoria, I have tried to steer clear of theoretic discussions and hypotheses unless they appeared to me necessary for the understanding of some individual processes.

This book, however, is addressed not only to the physician who in the stress of daily work has not the time to pay sufficient attention to the manifoldly disseminated literature and to follow up the progress exhibited in the same, but also to all those who, whether in teaching or in learning, are more familiar with this branch. The latter will find that this treatise does include no slavish leaning to "celebrated patterns," but nearly everywhere occupies a standpoint of its own acquired by long years' unbiased observation. Though the same may not always prove right, and as I do not doubt the never-resting work for research may overthrow to-morrow what appears to us as secure to-day, I acquiesce in the maxim: "In magnis voluisse satis!"

In the sense of these principles, I trust that I may have succeeded through this present treatise in further awakening and stimulating the interest for the diseases of the alimentary apparatus, the quite particular domain of the work and labour of the physician.

In conclusion, I desire to express my best thanks to Herrn Sanitätsrath Dr. S. Guttmann in Berlin for his manifold aid and practical hints in the preparing of this book.

THE AUTHOR.

Berlin, July, 1890.

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INTRODUCTION.

Diagnosis of diseases of the stomach, although a subject of considerable interest since the earliest rudiments of therapy, is one of the most difficult parts of clinical pathology. In the course of many centuries anatomy and physiology have experienced the greatest changes, which naturally have influenced the knowledge of the pathological disturbances of the digestive apparatus. It is the mission of modern medicine to select the truly useful from what, for some time, has been a mixture of erroneous and true, as well as from dogmatisms and fallacies.

Modern times are interlinked with the epoch-making evolution of physical methods, the practice of *percussion* and *auscultation,* by means of which our views concerning the location and size of the diseased stomach have undergone valuable changes and improvements. Yet, on the whole, the results of such efforts have remained far behind those obtained in the sphere of cardiac and pulmonary affections. The confusion which, till then, had existed in the knowledge of diseases was little changed; and up to the present time the fallacies of a theory founded exclusively upon physical symptomatology can distinctly be traced.

The introduction of the stomach-tube for diagnostic purposes by v. Leube, and of the stomach-pump for therapeutic purposes by Kussmaul, and the associated therapeutic results, are further landmarks in the history of diseases of the stomach. Through this, clinical procedure turned in a new direction, that of *functional diagnosis.* A large coterie of learned men have successfully associated themselves in the development of the fundamental principles established by v. Leube and Kussmaul, in consequence of which not a few views have undergone a change, which to-day can be considered as a permanent acquisition to our medical knowledge. It is true that what at first was a sanguine expectation, the diagnosis of the alimentary tract based upon chemical formulæ, has proved a fallacy; and we now know that even the stomach-tube does not answer all the questions of diagnosis, nor solve all the problems. But with increasing experience the functional method will maintain its acquired position *as an important, yes, indispensable, factor in the category of our methods of exploration.* Its value may perhaps be best compared to the significance of the examination of the urine in renal disease. To-day, no physician would want to diagnosticate a disease of the kidney on palpation alone, without reference to the composition of the urine, as, *vice versa,* the careful diagnostician would never estimate the examination of the urine as decisive in itself.

This view does not lack adherents nor antagonists. The latter claim that through the examination of the gastric contents, neither diagnosis nor therapy has acquired any great advantage. If my view is correct, it appears to me that this adverse decision rests upon too great a demand from the functional test. They demand decisive characteristics where the test can but contribute to the elucidation in diagnosis. Whoever does not recognise this process ought to prove how, in any other manner, chronic gastritides, insufficiency of the gastric musculature, neuroses, pyloric stenoses, beginning carcinoma, etc., can not only be suspected, but recognised, and how, on the other hand, as often enough occurs, a suspected disease of the stomach can be excluded. That such is not possible, except in exceptional cases, without functional investigation of the stomach, is proved by the confusion which has existed in the literature on the subject up to but a very few years ago.

Still more *incontestible* is the progress from a *therapeutic standpoint,* as is best evidenced by the fact that to-day the cure of diseases of the stomach is looked for, not in the stomach-bitters and so-called digestive remedies, but rather in an *individualised, rational way of living, in the broadest acceptation of the term.* Furthermore, the few remedies which the physician who stands at the height of science employs in diseases of the stomach are no longer tried one after the other, as used to be done in all seriousness, but rather upon a secured basis, the result of definite reflection.

A peculiar interest, hardly offered in an equal manner by any other branch of pathology, lies in the manifold and intricate *mutual relations between gastric and other diseases.* As a matter of course, the patient is often compelled and enabled to observe disturbances in the sphere of digestion very early and persistently, while the careful physician, meditating and observing with more critical eye, will consider them as of inferior importance in the symptomatology. How often does it not happen that a patient enters the physician's consulting room as a dyspeptic, and leaves it as a tabetic, nephritic, or diabetic? To bring light and elucidation into such apparently confused groups of diseases is the task of the thinking physician, and in the fact that rich experience and detailed knowledge are necessary for it, lies the authorisation to cultivate the *discipline of digestive diseases as a special branch, practically as well as scientifically.*

Just as the diseases of the nervous system have emancipated themselves from the maternal trunk of internal medicine, without having given up the connection with it, the pathology of the diseases of the digestive organs must—if it shall maintain its estate—not by any means lose the connection with the general practice of medicine.

CHAPTER I.

Preliminary Remarks on Topographic and Histologic Anatomy.*

The stomach is the beginning of a membranous tubular system, which continues at first in the form of coils (small intestine), and then passes in uniform loops (large intestine), to end finally in a descending portion below and externally (rectum). The stomach, the widest and most dilated portion of the alimentary canal, lies inserted between the œsophagus and small intestine. It is bounded above by the diaphragm and liver, below by the small intestine and the transverse colon, which forms a sort of cushion for it, and in front by the false ribs and the anterior abdominal wall; on the left it is bounded by the spleen, and on the right it reaches the median surface of the gall-bladder. Three-fourths of the stomach lie in the left half of the body, and the other fourth lies in the right side of the body and includes a portion of the fundus as well as the region of the pylorus. Under normal conditions the latter is covered by the left lobe of the liver, and therefore cannot be examined, as a rule, by inspection and palpation. The long axis of the stomach passes from above on the left in the front downward to the right in the back.

The pyloric region of the stomach (antrum pyloricum) is somewhat elevated in the median line, and approaches the right arch of the ribs. It has the shape of a conical sac, whose axis runs obliquely from the left forward and downward, and then backward and upward to the right. Posteriorly it lies upon the trunks of the portal vein and the hepatic artery.

The pylorus itself lies above, at the seventh or eighth rib and on a plane with the ensiform process, so that a line drawn downward between the right sternal and parasternal line strikes exactly the middle of the pylorus. This may, however, present decided variations under physiologic conditions.

The upper portion of the fundus of the stomach passes close to the diaphragm, from the left to the right, and is covered by the left lobe of the liver. The upper portion of the fundus is intimately united with the diaphragm by means of union between the serosa of the stomach and the peritoneal covering of the diaphragm. The highest point of the fundus lies in the mammillary line in the fourth intercostal space, near the fifth rib; from that point to the insertion of the seventh rib the fundus is covered by lung-tissue. The lower (anterior) portion of the fundus lies, when the stomach is full, against the thoracic wall; but when the organ is empty those structures are parted by either a flexure of the colon or a portion of the great omentum.

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* Use has been made of the text-books on anatomy by Henle, Quain-Hoffmann, Hyrtl, Gegenbaur, Rüdinger, as well as the work of Luschka on "The Position of the Abdominal Organs in the Human Being," Karlsruhe, 1873, and the article on "Stomach" in Eulenburg's Realencyclopædie, 1897, 3 Auflage, Bd. 14 (Klemensiewicz).

The cardiac portion of the stomach (pars abdominalis œsophagi, Luschka), that is, the part of the stomach situated between the mouth of the œsophagus and the true beginning of the fundus ventriculi, reaches as high as the sternal end of the sixth or seventh rib, the corresponding point posteriorly being the beginning of the eleventh dorsal vertebra. The cardia itself is inserted between the fundus and the small curvature, and lies from two to three centimetres below the diaphragm. Inasmuch as it is completely covered by the left lobe of the liver, cardiac growths are palpable only under exceptional circumstances (gastroptosis).

One portion of the posterior surface of the stomach is turned toward the posterior wall of the abdominal cavity, but touches it at no point, whereas the other portion is directed downward. The small curvature crosses the pancreas. From the median line onward the splenic artery runs parallel to the edge of the pancreas, on account of which a perforating ulcer of the posterior wall of the stomach may easily cause profuse hæmorrhages (Klemensiewicz).

Of great importance from a practical point of view is the direction in which the curved lines of the stomach run, that is, the greater and lesser curvatures of the stomach.

The greater curvature turns with its convexity toward the left hypochondrium and the anterior abdominal wall. In the upper portion of the fundus it is almost completely covered by lung-tissue and it does not begin to touch the abdominal wall until it reaches about the junction between the ninth and tenth ribs. From this point on there is but a slight curve until the median line is reached. From here on the curvature becomes more decided as it rises upward to the right, and passes in front of the median surface of the gall-bladder, beneath the liver, into the pylorus of the stomach. The lowest portion of the greater curvature is marked by a line drawn between the insertion of the ninth and tenth ribs and a point three to four centimetres above the umbilicus.

The lesser curvature, which is completely covered by the left lobe of the liver, and therefore inapproachable in the normal position by direct examination, travels at first with a slightly curved arch to the left from the spine and downward. Then, in the neighbourhood of the twelfth dorsal or first lumbar vertebra, it makes a short turn to the right of the median line, after which it runs fairly parallel to the greater curvature and forms a boundary to the pylorus.

The vertical position is considered to be the normal one of the stomach (see Fig. 1) by various recent authorities, especially Doyen[1] and Rosenfeld,[2] also v. Mikulicz and Kausch.[3]

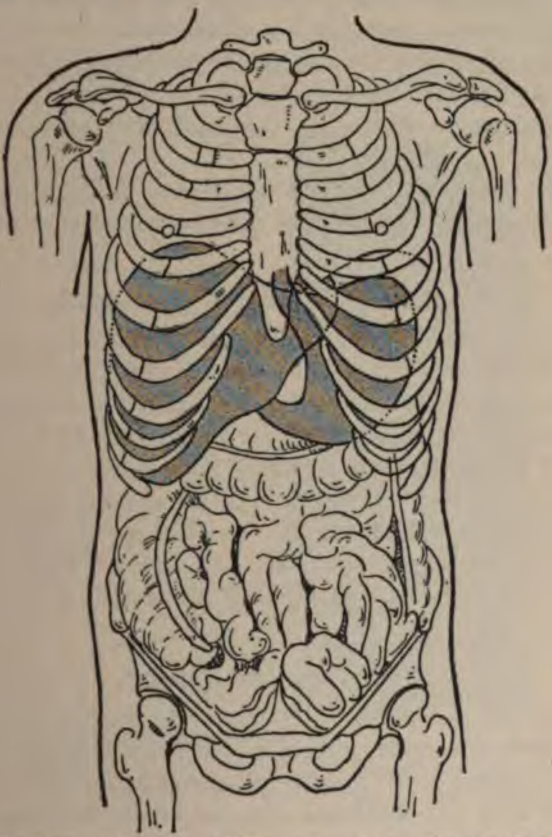

Fig. 1.—Position of the Normal Stomach. (According to G. Rosenfeld.)

According to Rosenfeld's investigations on the cadaver and on living subjects, the small curvature passes from the cardia to the left and downward. In the second third of its course it changes its direction and makes a curved or angular turn toward the right, to pass in front of the spine. The greater curvature corresponds in general to the accustomed teaching. The pylorus, in its described position on the right, often lies close to the spinal column in the region of the first and second lumbar vertebræ, but not rarely also in the left half of the body, so that the whole stomach, from the cardia to the pylorus, may lie to the left of the vertebral column. As Rosenfeld very properly stated, this position explains the fact that pyloric tumours may be palpated in the median line or to the left of it. In the same manner may be explained the tendency of ulcers to attack the lesser curvature, for the œsophagus empties its contents directly upon this point of the stomach.

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[1] Doyen, Traitement chirurgical des affections de l'estomac, Paris, 1895, S. 18.

[2] G. Rosenfeld, Centralblatt für innere Medicin, 1899, Bd. 20, No. 1; Zeitschr. f. klin. Medicin, 1899, Bd. 37, S. 81; Kongressverhandlungen für innere Medicin, 1899, S. 350; Münchener med. Wochenschrift, 1900, No. 35, S. 1204.

[3] v. Mikulicz und Kausch, Handbuch der prakt. Chirurgie von v. Bergmann; v. Bruns und v. Mikulicz, Separatabdruck, S. 93.

The contradiction between the recognised teachings of text-books on anatomy and the latter statement cannot be reconciled at present.

The Size and Capacity of the Stomach.

When the stomach is filled, its largest diameter measures from 25 to 30 centimetres; the diameter of the transverse section measures at its widest point 8 to 10 centimetres, but at the pylorus only 2 to 5 centimetres. When the organ is empty its longest diameter equals only 18 to 20 centimetres; the breadth, 7 to 8 centimetres when the folds of the pyloric mucous membrane lie closely together. The capacity of the stomach naturally varies considerably; according to Ewald[4] the smallest stomach holds 250 cubic centimetres, and the largest 1680 cubic centimetres. This author considers only a capacity of over 1600 to 1700 cubic centimetres as pathologic.

Fastening of the Stomach.

Under normal conditions only the lower portion of the fundus is movable, whereas the upper portion of the fundus, the pars pylorica and cardiaca, are immovable, the pylorus being slightly movable. The former is closely united with the peritoneal covering of the diaphragm so that the movements of the fundus correspond to those of the diaphragm. On the right the cardia is fastened by the gastro-hepatic ligament (lesser omentum), on account of which displacement of the cardia is impossible. The pyloric end is fastened by means of the duodenum, with which it is directly connected to the side of the vertebræ. The absence of a true ligamentous attachment allows the pylorus certain excursions, especially downward, so that a marked descent of the pylorus, even into the pelvis, is not rarely observed in the cadaver. The great omentum extends from the greater curvature downward toward the pelvis, and covers the loops of the small intestines like an apron (Hyrtl), after which it folds on itself and passes upward to end in the transverse colon. The portion of this membrane lying between the stomach and transverse colon is called the gastro-colic ligament. In case of excessive distention of the stomach, the fundus, notwithstanding this attachment, may, as experience shows, stretch downward to various degrees. A ligamentous connection exists likewise between the stomach and spleen, the *gastro-splenic ligament,* and also between the stomach and the pancreas, the *gastro-pancreatic ligament.*

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[4] Ewald, Klinik der Verdauungskrankheiten, Theil II, 3 Aufl., p. 86.

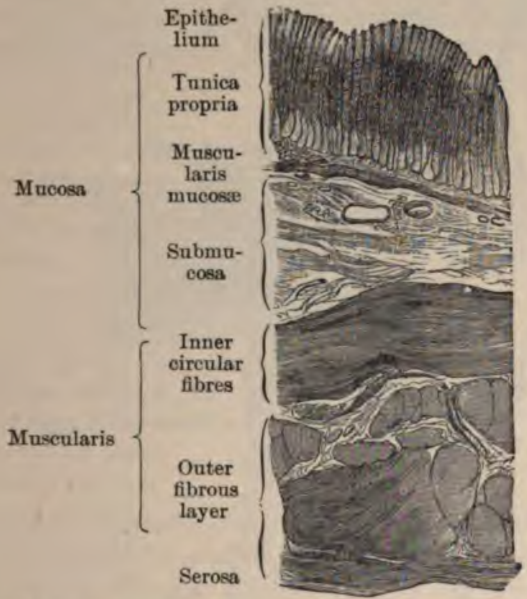

Fig. 2.—Vertical Section Through the Human Stomach-Wall. The glands are crowded so closely together in the tunica propria that their structure can only be seen at the base of the glands near the muscular coat. (From Stöhr.)

Histologic Anatomy.*

The *inner surface* of the stomach is divided into a large number of planes by numerous lattice-like folds, within which small projections are noted which are designated as mammillæ. This uneven appearance is termed, especially when it is marked, as *Etat mammeloné.* By means of the folds it is possible to provide for a very rich glandular apparatus. They may be obliterated by exerting tension. The openings of the stomach, the cardiac and pyloric antra, are distinguished by elevations above the rest of the mucous membrane. The eminence caused by the former is represented by a reddish-gray, smooth surface, which forms a marked contrast with the serrated and pale border of the œsophageal mucous membrane. The pyloric opening is marked off from the duodenum by a duplication of the mucous membrane (*pyloric valve*). A hardened specimen of the stomach-wall, which measures 2 to 3 millimetres in thickness, may be divided into four layers: the mucous, submucous, muscular, and serous coats (see Fig. 2). The *mucous membrane* is composed of epithelium, the tunica propria, and muscular layer.

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* The above-cited literature has been employed, as well as the excellent article by Heidenhain in Hermann's Handb. der Physiologie, Bd. 5; Stöhr, Lehrbuch der Histologie, 1889, 3 Aufl., A. Oppel, Lehrbuch der vergleichenden mikrosk. Anatomie der Wirbelthiere, 1 Theil; Der Magen, Jena, 1896.

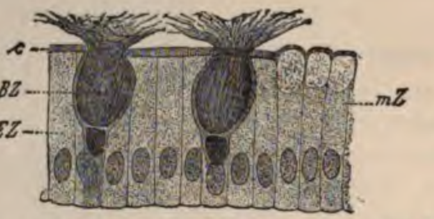

Fig. 3.—Heterotopic Intestinal Epithelium from the Human Cardia. Enlarged 560 times. *BZ*, Beaker cells. *EZ*, Epithelial cells with a cuticular border *c*. *mZ*, The same with the formation of superficial vacuoles. (According to J. Schaffer.)

The *epithelium*, which lines the whole surface of the gastric mucous membrane, and which is invaginated to form little fossæ or pits, is a simple cylindrical epithelium, which contains a mucoid substance. The production of mucus is accompanied by certain alterations in the protoplasm, which are sharply distinguished from each other by changes in color. Accordingly we may distinguish between an upper mucous portion and a lower (basal) protoplasmic portion which contains an oval nucleus. Aside from the above-mentioned epithelial cells, true intestinal epithelium with its peculiar cuticular border may also be found, as has been shown by recent investigations. (Fig. 3.) In such areas beaker-cells are also demonstrable, which present all the characteristics of those cells in the small intestine. One was inclined formerly to consider these structures as pathologic (obstruction of the cells with mucus), but the investigations of Ad. Schmidt[5] and P. Hári[6] prove them to be a normal formation, which may be looked upon as displaced intestinal epithelium.

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[5] Ad. Schmidt, Deutsche medicinische Wochenschrift, 1895, No. 19, S. 300, and Virchow's Archiv, 1896, Bd. 43, S. 477.

[6] Paul Hári, Archiv für mikroskopische Anatomie, 1901, Bd. 58, P. 678.

They are almost exclusively found, according to Hári, in the pyloric region and the intermedia, but under certain circumstances they are also met with at a greater distance from the pylorus. They may, however, be absent from the immediate neighbourhood of the pylorus. The mucous membrane surrounding the region in which they are lodged may be free of all pathologic change, but, on the other hand, it may present the severest grade of interstitial and parenchymatous glandular disease.

The *tunica propria* is composed of a fibrillar and in some places reticular connective tissue, between which small or large collections of leucocytes are seen. Elsewhere it forms the narrow interstitial substance between the glands. This interstitial substance is better developed only in the region of the pylorus. The gastric glands form the essential element of the tunica propria; these are distinguished as the glands of the fundus and those of the pylorus. The former, whose function is chiefly the secretion of acids and ferments, are single or forked tubes, which empty into the so-called gastric (Donders) or glandular follicles (Heidenhain)

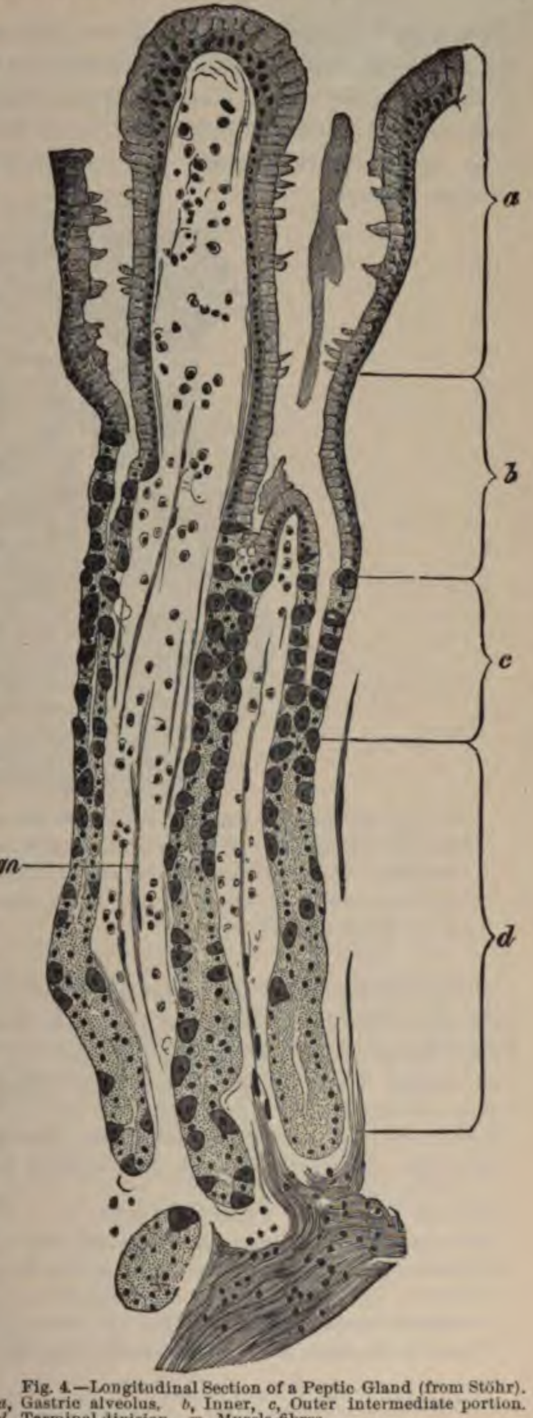

Fig. 4.—Longitudinal Section of a Peptic Gland (from Stöhr). *a*, Gastric alveolus. *b*, Inner, *c*, Outer intermediate portion. *d*, Terminal division. *m*, Muscle-fibres.

(Figs. 4 and 5). The portion of the gland next to the gastric follicle is the narrowest, and is called the glandular neck; then comes a thicker portion, the body of the gland, which is followed by a somewhat narrower portion, the fundus. Rollet divides the gland into the gastric alveolus, the inner intermediate portion, the outer intermediate portion, and the terminal portion.

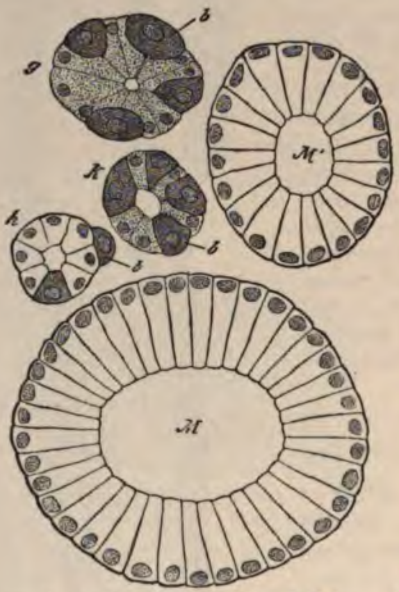

Fig. 5.—Cross-sections of a Gastric Gland Taken at Different Levels. Enlarged 400 times. *M*, Gastric alveolus near the surface. *M'*, Somewhat lower, near the division of the gland. *h*, Lumen of the glandular neck. *k*, Glandular neck. *g*, Body of the gland, near the base. *b*, Border cells. (From Kölliker-v. Ebner.)

Our knowledge of the histologic construction of the gastric glands has been considerably advanced by the more recent investigations of Heidenhain,[7] Rollett,[8] Jukes,[9] Edinger,[10] Stöhr,[11] Kupffer,[12] Sachs,[13] Bonnet,[14] and others. According to them the glands of the fundus possess two varieties of cells, the chief and the parietal or border cells. The former are pale, cuboidal or cylindrical cells, with granular protoplasm which possesses a spherical nucleus. They stain less readily, according to Krause,[15] with hæmatoxyline, carmine, aniline, and potassium bichromate, than the cylindrical cells of the excretory ducts or the parietal cells.

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[7] Heidenhain, Archiv für mikroskopische Anatomie, 1870, Bd. 6.

[8] Rollett, Untersuchungen aus dem Institut f. Physiol. u. Histologie zu Graz, 1870, Heft 2.

[9] Jukes, Inaug-Diss., Göttingen, 1871.

[10] Edinger, Archiv f. mikrosk. Anat., 1880, Bd. 17.

[11] Stöhr, Arch. f. mikrosk. Anat., 1882, Bd. 20.

[12] Kupffer, Epithel und Drüsen des menschlichen Magens, München, 1883.

[13] Sachs, Arch. f. experim. Pathol., 1887-1888, Bd. 22 et 24.

[14] Bonnet, Deutsche medicinische Wochenschrift, 1893, No. 18, S. 430.

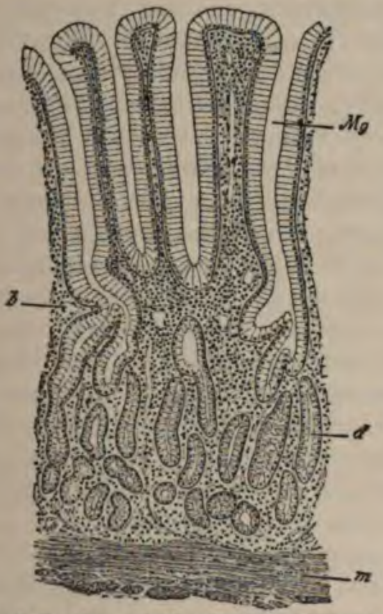

Fig. 6.—Vertical Section of the Pyloric Mucous Membrane of the Stomach. Enlarged 85 times. *Mg*, Gastric follicles. *b*, Blood-vessels in the mucous membrane. *d*, Glandular tubules. *m*, Muscularis mucosæ. (From Kölliker-v. Ebner.)

When treated with 0.5 to 5 per cent. solution of acetic acid and 0.02 to 0.05 per cent. solution of nitric acid, the border cells become clear, whereas the protoplasm of the chief cells becomes cloudy. The chief cells are more strongly stained with the aniline dyes during digestion than during the fasting period. The border cells are large, polynuclear structures, which are usually round, but may occasionally be triangular. The distribution of the chief and border cells is such that the latter predominate especially in the neck and body of the glands, whereas the base possesses only a few border cells.* In certain regions of the fundus these cells extend more or less beyond the even line of the remaining cells, and form a protuberance, but in other sections, especially toward the base of the gland, they lie in the periphery along the lumen of the duct (Fig. 5).

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* The border cells are claimed to be absent at the base of the glands, according to Heidenhain, whose view is, however, disputed by Stöhr, Kupffer, and others, including myself.

[15] Krause, Allgem. u. mikrosk. Anat., Bd. 1, Handbuch d. menschlichen Anat., Hannover, 1876; Cit. nach Oppel, *l.c.* S. 471.

Opinion is divided as to the significance of the chief and border cells. Heidenhain,[16] Grützner,[17] v. Swiecicki,[18] and Sehrwald[19] believe that the chief cells produce the ferments and that the border cells secrete the acids; whereas S. Fränkel,[20] F. Klug,[21] and Contejean[22] found the function of the border-cells to consist in the elaboration of hydrochloric acid and pepsine. Fränkel and Contejean furthermore found, in accordance with the earlier investigations of Edinger, the reaction of the whole gastric mucous membrane, including that of the pyloric end, to be undoubtedly acid, which is contrary to the findings of Ebstein, Klemensiewicz, and Heidenhain, who determined that the pyloric secretion was alkaline in reaction. Vacuoles have been observed in the border cells by Heidenhain, Stöhr, Sachs, Stintzing, Hamburger, and Bonnet. All of these authors, excepting the latter, found the vacuoles near the nucleus, whereas Bonnet found them partly near by and partly at the site of the nucleus. The explanation of this remarkable phenomenon is uncertain, but it appears likely that it is a manifestation of the process of secretion. Bonnet has furthermore determined that a portion of the nuclei in the polynuclear border cells, which are rich in chromatin, belongs to leucocytes. The latter may be observed in the border cells at all stages of invasion.

The *glands of the pylorus* (Fig. 6) also consist of coils of tubules, which may occur singly or divided into many branches. Here, as in the case of the gastric glands, the gastric follicle leads at first to the neck, which in turn is followed by the body and then the fundus of the gland. The gastric follicle is lined with epithelium, which is of the character of the superficial epithelium, whereas the true glandular cells possess the characteristics of the chief cells of the glands of the fundus. Still other disseminated cells are noted, which in their structure and reaction tend to resemble the border cells (Nussbaum's cells). These, however, have more recently been looked upon by Stöhr and Hamburger as glandular cells which have become altered by compression from neighbouring cells.

The lab glands *in the bordering zone between the pylorus and fundus* of the stomach differ somewhat in appearance, inasmuch as they are shorter and more spiral in shape. In the neighbourhood of the cardia the border cells may be completely absent, according to Kupffer.[23] The single or forked gastric alveoli, which extend to one-half of the thickness of the mucous membrane, pass into wide, partly shorter, and partly longer spiral glandular tubules, which are paved only with a uniformly-low epithelium. Aside from the above-mentioned glands, we also note simple *mucus glands*, that is, glands, including lymphatic glands, which are wholly lined with cylindrical epithelium. The lymphatic glands lie as little nodules in the tissue of the mucous membrane, and when intensely swollen may press the glandular tubules apart.

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[16] Heidenhain, Schulze's Archiv für mikroskopische Anatomie, 1870, Bd. 6, S. 368.

[17] Grützner, Archiv. f. d. ges. Physiologie, 1879, Bd. 20, S. 410.

[18] v. Swiecicki, Archiv f. d. ges. Physiologie, 1876, Bd. 13, S. 452.

[19] Sehrwald, Münch. med. Wochenschr., 1888, No. 11.

[20] S. Fränkel, Pflüger's Archiv, 1890, Bd. 48, S. 63.

[21] F. Klug, Ungar. Archiv f. Medicin, 1892, Bd. I, S. 35.

[22] Contejean, Archives de Physiologie 1892, Serie V., Bd. IV. 3, S. 554-561.

During the process of digestion the glandular elements change their appearance in a characteristic manner. The border cells become larger and swollen, but lessen in size as the end of digestion approaches. The chief cells likewise undergo enlargement and become darker and cloudy, but do not assume their former appearance and size until several hours after digestion.

The so-called *fatty granular cells* (*Mastzellen*) occur, according to Stintzing,[24] in large numbers in the fundus as well as in the pylorus. In the healthy stomach they are never seen outside of the connective tissue, but in pathologic conditions they appear between the tunica propria and the glandular cells. They may penetrate between two glandular cells and reach as far as the lumen. This process should not be confused with the passage of the leucocytes through the superficial epithelium.

The *connective tissue* of the human stomach is very scanty in the fundus. The glands lie closely together, and are separated only here and there by a little connective tissue and muscle-fibres arising from the muscularis mucosæ, or by blood-vessels. The pylorus is more richly supplied with connective tissue and smooth musculature, and exhibits a decided leucocytic infiltration and numerous lymphatic nodules (Bonnet).

The *muscularis mucosæ* is composed of two or three layers of smooth muscle-bundles, extending in various directions. Offshoots from the musculature pass upward to the glandular portion of the mucous membrane.

The *submucosa* consists of loose connective-tissue bundles alone, or combined with elastic fibres, forming a support for the mucosa. Smaller or greater collections of fat-cells are found occasionally in the connective-tissue mass.

The *muscularis ventriculi* presents in the pylorus two distinctly separate fibrous systems (Fig. 2): an inner circular and an outer longitudinal layer. In the other portions of the stomach the admixture of muscle-fibres from the œsophagus presents a most complicated picture, so that on cross-section the musculature exhibits a most varied arrangement. It is, however, of no practical interest to describe this subject here in detail.

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[23] Kupffer, *l.c.*

[24] Stintzing, Münch. med. Wochenschr., 1889, No. 8, S. 121.

Blood-vessels and Nerves of the Stomach.

The *arteries* of the stomach arise from the cœliac artery. Of its branches, the left coronary artery, which arises directly from the cœliac, and the right coronary artery, which arises from the hepatic artery, supply the lesser curvature. These two vessels form the superior ventricular arch. The greater curvature is likewise supplied by a branch from the hepatic artery, the left coronary gastric artery, which is a branch from the splenic artery to the inferior ventricular arch.

The *veins* run, as a rule, parallel to the course of the arteries. Those arising from the left coronary artery empty into the splenic vein; those accompanying the right coronary artery end in the mesenteric or portal vein, into which, as a rule, the superior coronary vein also empties. An independent venous trunk is found at the pylorus, the pyloric vein, which empties either into the inferior coronary vein or directly into the portal vein. The vessels pass from the serosa through the muscularis to form a plexus in the submucosa. This plexus spreads out over a level plane, and emits fine capillaries, which, surrounding the glandular tubules, continue upwards to construct a second plexus at the mouth of the gland. It, in turn, gives off other fine capillaries, which run in a circle around the exit. Minute veins arise from the capillaries, which, in a manner similar to the arteries, run downward to build a venous network in the tunica propria.

The *lymphatic vessels* form large, tube-like spaces around the glandular tubules, which are bounded on the one side by the true membrane of the glands and on the other side by the endothelium of the interstitial connective-tissue (Lowén). Lymph (chyle) capillaries, running between the individual tubules, connect with a widely-meshed network in the submucosa. The lymph-vessels, arising therefrom and supplied with valves, spread throughout the muscular coat; they also receive the many lymph-capillaries existing in the muscular strata. The lymph-vessels run beneath the serosa to the mesentery, between the layers of which they continue their course.

The *nerves* of the stomach originate partly in the vagus and partly in the solar plexus. The fibres, which are mostly non-medullated, form a network in the serosa, from which they penetrate the muscularis to build an extensive plexus between the longitudinal and circular layers, called the mesenteric plexus (plexus Auerbachii). Numerous multipolar ganglion-cells lie at the nodal points of this plexus. Nerve-fibres extend from this plexus to end either in the muscle-fibres or, penetrating into the submucosa, form a second plexus, called Meissner's plexus. Small fibres run from this plexus between the glands to the glandular cells.

CHAPTER II.

Preliminary Remarks on Physiology and Chemistry.

The study of the physiologic and chemical processes of digestion varies according to whether the point of view is that of a physician, a physiologist, or a chemist. The two latter are concerned only in the process itself, whereas a physician must draw certain conclusions from the normal process which will aid him in the treatment of his patients. The physiologist studies the organ itself, separated from the rest of the digestive tract; but the physician considers the gastric digestion as only a partial phenomenon of digestion, and therefore includes also the oral and intestinal digestion. The following discussion will only cover the cardinal points of the processes of digestion; for a more detailed consideration of the subject, reference should be made to text-books on physiologic chemistry (Hammarsten, Bunge, Neumeister, Gamgee, and others).

That digestion begins with the dividing and masticating of the morsels and with the swallowing of liquids is common knowledge. All physicians are familiar with the fact that the food in the mouth is intimately mixed with *saliva* (salivation), which continues its activity in the stomach with the production of important changes, and that the morsel becomes simultaneously thinner and lubricated. We know, moreover, that carious teeth, putrefactive oral processes, and disease of the salivary glands have a decided influence upon digestion. These disturbances are largely due to bacteria, our knowledge of which was greatly advanced by the investigations of recent years. We are especially indebted to the noteworthy investigations of W. D. Miller[1] on the bacteria of the mouth and their relation to the digestive processes, to a consideration of which we will return later. Miller found two groups of organisms in the mouth, as well as in the stomach, which produced fermentation. The one variety decomposes carbohydrates, with formation of acid substances, and the other type causes decomposition of proteids, with formation of alkaline products. We observe either one or both varieties of fermentation, depending upon the presence or predominance of one or the other form of food. Miller has furthermore demonstrated that the action of microbes upon the proteids led constantly to putrefactive changes, with formation of sulphuretted hydrogen, ammonia, carbonic acid, etc., and a large number of bases (ptomaines). There can be no doubt, therefore, that a solution, or rather decomposition, of the proteids occurs under those circumstances.

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[1] Miller, Die Mikroorganismen der Mundhöhle, 2 Auflage, Leipzig, 1892, G. Thieme.

That this process is comparable to the peptonising property of the well-known gastric and intestinal enzymes is, however, out of the question, a fact which was also emphasised by Miller. We have, therefore, to look upon micro-organisms which decompose the proteids as injurious to the digestive cycle and the general health of the body. These investigations have considerably advanced our knowledge of the reciprocal relations existing between the digestive processes of the mouth and of the stomach, well-known even to the earlier physicians (see below). The most important secretion of the oral cavity is the *saliva,* the significance of which in the digestion of food will be discussed in detail.

The Saliva.

The *saliva* is a mixture of the secretions from the salivary and mucus glands, which consists of a viscid and tenacious opalescent fluid. The quantity of saliva secreted during twenty-four hours equals, according to Bidder and Schmidt,[2] 1400 to 1500 grammes; according to Fr. Krüger,[3] 250 to 300 grammes; according to W. G. A. Robertson,[4] 331 to 407 grammes. The reaction is strongly alkaline, provided an indicator (methyl-orange) is employed which remains unaffected by CO_2; whereas the reaction is found to be decidedly acid if phenolphthalein be used, which is very sensitive to CO_2 (Dieminger,[5] M. Cohn[6]). According to Chittenden and Ely, the alkalinity equals a solution of 0.8 per mille Na_2CO_3. The degree of the alkalinity presents certain variations in the course of a day.

Its specific gravity equals 1002 to 1009, usually from 1003 to 1004. The amount of solid matter equals 5 to 10 per cent., and consists of epithelium, mucus, ptyaline, albumen, and salts. A constituent peculiar to saliva is mucin. The analysis given in the table on page 17 (by Hammarsten) presents the average constituency of the human saliva. The figures are referable to 1000 parts.

The mixed saliva also contains an acid combined with potassium thiocyanate, which has not yet been isolated and which appears to be practically of secondary importance. This constituent may be demonstrated by acidulating the saliva with hydrochloric acid and adding, drop by drop, a

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[2] Bidder and Schmidt, Die Verdauungssäfte und der Stoffwechsel, Mitau and Leipzig, 1852.

[3] Fr. Krüger, Zeitschrift für Biologie, 1897, Bd. 37, S. 6-24.

[4] W. G. A. Robertson, Journal of Anatomy and Physiology, 1898, Bd. 32, S. 615.

[5] Dieminger, Inaug-Diss., Würzburg, 1898.

[6] M. Cohn, Deutsche medicinische Wochenschrift, 1900, No. 4.

	Berzelius	Jakubowitch	Frerichs	Friedmann and Gmelin	Herter	Lehmann	Hammerbacher
Water	992.9	995.16	994.1	988.3	994.7	. . . . .	994.2
Solid Material . .	7.1	4.84	5.9	11.7	5.3	3.5-8.4	5.8
Mucus and Epithelium . . .	1.4	1.62	2.13	. . . .	. .	(Filtered Saliva) . . .	2.2
Soluble Organic Substances . . .	3.8	1.34	1.42	. . . .	3.27	. . .	1.4
Potassium Thiocyanate	. .	0.06	0.10	. . . .	. .	0.064—0.09	0.04
Salts . .	1.9	1.82	2.19		1.30		2.2

weak solution of the chloride of iron, which produces a dark-red or Burgundy-red colour (formation of ferri-thiocyanate). Or add a little hydriodic acid to the saliva, which is reduced by means of formation of thiocyanate and free iodine, the latter being easily detected by means of starch-paste (reaction of Solera). The amount of potassium thiocyanate is frequently increased in cases of chronic pharyngitis and of abnormal formation of mucus, also in association with vomitus matutinus, and, according to Fr. Krüger, also with tobacco-smoking; its amount is lessened in cachexia and severe diseases of long duration (Jul. Grober[7]).

According to Schönbein's[8] observation, *nitrous acid* is also present in the saliva. If a solution of potassium iodide in starch-paste is added to the saliva which has been acidulated with sulphuric acid, the formation of iodide starch will frequently be noted.

The principal action of the saliva is diastatic, which depends upon the presence of an extremely tenacious and active enzyme, called *ptyaline,* or better, salivary diastase. It converts all the starches, as well as glycogen, into sugar. Ptyaline acts in a slightly alkaline, neutral, or extremely slightly acid medium. It is most energetic, according to Chittenden and Smith, in neutral or even better in a slightly acid reaction. Ptyaline occurs not only in adults, but also in newly-born infants. Zweifel[9] claims that in the new-born, ptyaline is found only in the parotid and not in the submaxillary gland. It is supposed not to appear in the latter until two months after birth.

Starch is converted, by means of the salivary diastase, into *maltose*

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[7] **Jul. Grober, Deutsches Archiv für klinische Medicin, 1901, Bd. 69, S. 243.**

[8] **Schönbein, Journal für praktische Chemie, Bd. 86, S. 451.**

[9] **Zweifel, Untersuchungen über den Verdauungsapparat der Neugeborenen, Berlin, 1874.**

(Dubrunfaut,[10] O'Sullivan,[11] Musculus and v. Mering,[12] Brown and Heron[13]), or *isomaltose* (E. Külz and J. Vogel,[14] K. Hamburger[15]), and minute quantities of dextrose. The dextrose appears to be but the product of inversion of the maltose by glucase (Tebb, Röhmann and Hamburger[16]). The conversion occurs more rapidly in case of cooked (starch-paste) than in raw starch, yet the saccharification of the latter, as I have discovered,[17] and as one may easily determine, occurs in a few minutes and is by no means slower than through action of the pancreatic diastase. The action of the saliva upon the food is almost immediate, so that reducing substances may already be found in the mass after a few seconds. Nevertheless, several intermediate stages are distinguished before complete saccharification occurs. These have been carefully studied by Brücke[18] and may be traced as follows:

1. The diastase causes liquefaction of the starch, which, in contradistinction to the starch-paste, is a true solution. This product, which is termed *amidulin* or *amylodextrine,* still gives a distinct blue colour when treated with a dilute solution of iodine in iodide of potassium (pure iodine 1.0, potassium iodide 2.0, aqua destillata 100.0).

2. The colour produced by the addition of the iodine solution changes gradually and becomes more violet-blue, violet, red-violet, red- or mahogany-brown. This modification of starch is designated *erythrodextrine.*

3. As the action of the diastase increases, the violet or brown colour gradually fades, until finally the addition of iodine shows a colourless dextrine, which is termed *achroödextrine.* While soluble starches may be precipitated by tannic acid and alcohol, the above-mentioned varieties of dextrine are precipitated only by alcohol. Herzfeld[19] claims that still another product occurs between achroödextrine and maltose, called maltodextrine, which closely resembles maltose, but which is unfermentable, according to Brown and Heron. The specific rotation of maltodextrine is represented by $(\alpha)\ D = 174.5°$.

The end product of the salivary diastase is *maltose or ptyalose* (Nasse), $C_{12}H_{22}O_{11} + H_2O$.

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[10] Dubrunfaut, Ann. Ch. Phys., Ser. 3, Bd. 21, S. 178; Cit. nach Gamgee, Die physiologische Chemie der Verdauung, Deutsche Ausgabe, 1897, S. 35.

[11] O'Sullivan, Journal of the Chemical Society, Ser. 2, Bd. 10, S. 579; Cit. nach Gamgee.

[12] Musculus and v. Mering, Zeitschr. f. physiol. Chemie, 1878 u. 1879, Bd. 2.

[13] Brown and Heron, Liebig's Annalen, Bd. 199 und 204.

[14] E. Külz and J. Vogel, Zeitschrift für Biologie, 1895, Bd. 31, S. 108.

[15] K. Hamburger, Pflüger's Archiv, 1895, Bd. 60, S. 543-597.

[16] Cit. nach Hammarsten, Lehrbuch der physiol. Chemie, 4 Aufl., S. 256.

[17] Boas, Zeitschrift für klinische Medicin, 1890, Bd. 17, Heft 1 und 2.

[18] Brücke, Wiener akademische Sitzungberichte, April, 1872.

[19] Herzfeld, Berichte der deutsch. chemischen Gesellschaft, Bd. 12, S. 2120.

Maltose, when isolated, consists of white crusts, composed of fine needles which are easily soluble in water as well as in ethyl- and methyl-alcohol, but somewhat less so in the first than is dextrose. Maltose possesses the specific rotation (α) D = 150.4 (Brown and Heron), therefore almost thrice that of dextrose, (α) = 52.5. Maltose possesses less reducing power for Fehling's and similar solutions than dextrose, it being able to precipitate only two-thirds of the amount of cuprous oxide precipitated by the latter. The exact relation between them, according to Brown and Heron, is 60.8:100. When an observation-tube 200 millimetres long is employed, every degree of rotation read off at 17.5° C = 0.362 maltose in 100 cubic centimetres. The reducing power of maltose is increased by treating it with dilute hydrochloric or sulphuric acid, and it is gradually converted into *dextrose*. According to Soxhlet, 1 cubic centimetre of Fehling's solution equals 7.78 milligrammes maltose in a 1 per cent. solution, provided the former is undiluted, and 7.4 milligrammes if it is diluted. Maltose in solution is directly fermentable by means of yeast. Dextrose is also distinguished from maltose by the fact that the former reduces Barfoed's reagent,* whereas the latter does not. Dextrose is furthermore differentiated from maltose by the fact that the glucosazon is almost insoluble in water, whereas the maltosazon is soluble in hot water.

The following table serves to demonstrate more clearly the different products formed during the conversion of starch:—

1. Soluble Starches (Amylodextrine, Amidulin)			Stains blue with iodine; precipitated by tannic acid and alcohol.
2. Varieties of Dextrine	Erythrodextrine	Iodine stains it violet to mahogany-brown colour	Precipitated in solution by alcohol and ether, but not with tannic acid. It does not reduce Fehling's solution, nor does the addition of yeast cause fermentation.
	Achroödextrine Maltodextrine	Iodine does not colour them	
3. Maltose			Soluble in alcohol, insoluble in ether, reduces Fehling's solution but *not* Barfoed's reagent. Yeast causes fermentation. Maltosazon is soluble in hot water.
4. Dextrose			Insoluble in alcohol and ether, somewhat soluble in diluted alcohol; Fehling's as well as Barfoed's solutions are reduced. Easily fermentable with yeast. Glucosazon is insoluble in water.

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* Barfoed's reagent consists of a solution of 1 part of acetate of copper in 15 parts water to 200 cubic centimetres; to this solution, 5 cubic centimetres of acetic acid (38 per cent.) are added.

Glycogen is decomposed by the salivary diastase, and likewise passes through several intermediary stages before it is converted into maltose and dextrose. If glycogen is added to filtered saliva, the brownish-red colour which results when that substance is treated with Lugol's solution disappears. A reducing substance gradually develops which is at first probably maltose, and later both maltose and dextrose.

The Physiologic Significance of Saliva.

The first action of saliva is mechanical, and consists in covering the food-bolus with mucus, which acts as a lubricant and thus assists it in its downward passage. Therefore, swallowing is made more difficult and delayed whenever an insufficient amount of saliva is excreted. Saliva also dissolves easily-soluble bodies like salt, sugar, etc. The important function of starch-digestion, which is continued to a certain extent in the stomach, begins in the mouth. The conversion of starch into sugar is of the highest importance to nutrition. Starch belongs to the class of colloidal substances which are diffused through animal membranes with difficulty. Sugar, on the other hand, belongs to the crystalloid products, which, in solution, pass through membranes with facility, and are therefore easily taken up by the blood and lymph. Finally, the large quantities of water which are swallowed with the saliva are not without significance to the body economy. "The animal organism possesses in saliva a powerful means for the production of a continuous stream between the intestinal canal and the blood during digestion, which carries with it the dissolved or finely divided food-products" (Hammarsten).

The Function of the Saliva in the Stomach.

To judge from the rapidity of the diastatic action of saliva, one might expect the complete saccharification of the carbohydrates would occur in a short time. That this, however, is not true may be easily determined by examination of the gastric contents in healthy subjects after the administration of an amylaceous diet. The starches may be found present after an interval of several hours, by testing with the iodine solution for the violet colour.

The action of the saliva becomes restricted by an increasing secretion of HCl, a fact which was first proven by van den Velden,[20] and then by Ellenberger and Hofmeister,[21] Ewald and myself.[22] He demonstrated that small quantities of acids limited the action of the salivary diastase, and that large quantities completely destroyed it. The following small table gives the percentages concerned, which were determined upon by Ewald and myself, and which coincide fairly accurately with the figures given by other investigators (Hammarsten, Chittenden and Griswold, Nylén, Langley and Eves, and others):

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[20] van den Velden, Deutsches Archiv für klin. Medicin, 1880, Bd. 25, S. 105.

[21] Ellenberger and Hofmeister, Archiv f. wissenschaftl. u. praktische Thierheilkunde, 1886, Bd. 12, S. 332.

[22] Ewald and Boas, Virchow's Archiv, 1886, Bd. 104, S. 271.

The action of the saliva becomes

	Restricted	by	Destroyed
Hydrochloric acid	**0.07 %**		**0.12 %**
Lactic acid	0.1 %		0.15 %
Butyric acid	0.2 %		0.4—0.5 %
Acetic acid			

The restriction of amylolysis is easily explained by the fact that under normal conditions the amount of hydrochloric acid equals 0.2 per cent., which may even become higher. Thus this unfavourable influence of the strong superacidity upon the process of saccharification is readily explained; whereas, on the other hand, when the diastatic activity is unrestricted, products of fermentation may develop to an excessive degree, a subject to which reference will be made later. This leads to the important question which does not seem hitherto to have received its due consideration, either from the clinical or physiologic standpoint, namely, whether an excess of acid destroys the diastatic ferment permanently or only facultatively; in other words, whether the diastase becomes active again when less acid is secreted or when it is neutralised with alkalies. I have attempted to solve this problem, and came to the conclusion that ptyaline again becomes active after alkalisation or after a decrease in the secretion of the acid. I was able, for instance, by means of alkalisation with a soda solution, to observe a return to activity of the diastatic ferment in a starch-mixture in which the saliva had been restricted for one hour by the presence of 0.15 per cent. of HCl. It follows, therefore, that saccharification probably again becomes active in the later stages of gastric digestion, when the acid production is diminished. A few authors, amongst whom van den Velden[23] was the first, have distinguished, on the above grounds, between two stages of digestion, namely, an *amylolytic* and a *proteolytic*. This is correct in so far that the amylaceous digestion predominates at the beginning when HCl is absent, but that the proteid digestion, which cannot dispense with free hydrochloric acid, is relatively insignificant. Nevertheless the products of proteolysis are plainly demonstrable in this stage.

Aside from its diastatic action, saliva seems to have a peculiar influence upon gastric digestion, which has received especial study from Sticker[24] and Biernacki.[25] The former claims that deficient salivary activity is followed by an arrest or diminution of HCl secretion. Biernacki, who made further investigations, showed that it was not the saliva itself, but its combination with the food in the mouth, which favoured digestion. A. Schuld[26] obtained the same results as Biernacki.

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[23] van den Velden, *l. c.*

The Gastric Juice.

The gastric juice in the pure state is a clear, almost colourless, fluid, which is acid in reaction and possesses a specific gravity of 1002 to 1003. It has a stale, sour taste. Its solid constituents equal only 0.56 per cent. The amount secreted has been variously estimated; thus Beaumont found it to equal about 180 grammes, and Grünewald 1580. According to the more recent investigations, the latter is more nearly correct than the former.

We must thank the genial Russian investigator, J. P. Pawlow,* and his pupils for a number of new and fundamental facts concerning the physiology of gastric secretion. The methods of Pawlow are distinguished from those of former investigators, especially of Heidenhain, by the fact that he was the first to be able to study conveniently the gastric secretion in the dog after the introduction of certain foods, and then determine the influence of the so-called sham-feeding upon the secretion of the gastric juice. Pawlow was thus likewise able to isolate the pancreatic juice and to observe the correlation between gastric and pancreatic secretion. In order to study the secretion of the gastric juice and its characteristics during the act of digestion, Pawlow constructed a small blind pouch out of a portion of the stomach, which was separated from that organ itself by a septum of mucous membrane, and which communicated externally by means of a fistula. The innervation of the blind sac remained wholly intact, so that upon the introduction of food into the stomach, the blind pouch portrayed a true and accurate picture of the secretory activity of the stomach without interference from the food.

The following conclusions were drawn: The gastric glands do not begin to secrete until food is introduced; the amount of the gastric juice is proportionate to that of the food. The secreting stomach is constantly acid in reaction, and the more rapidly the gastric juice appears the greater is the amount of acid secreted. The total percentage of acidity is furthermore dependent, aside from the amount of gastric juice present, upon neutralisation with mucus, etc. The activity of the gastric glands, after the ingestion of single components of food (like meat, bread, milk), proceeds with an extraordinary exactness, both with reference to the amount secreted and the ferments contained in the juice; this process may be recorded in the form of a distinct curve. Pawlow proved, furthermore, that the gastric juice which is secreted after bread-diet possesses the greatest digestive power; next in digestive strength comes the gastric juice following meat-diet, whereas the least digestive strength is possessed by the gastric secretion which follows the ingestion of milk-diet. The total acidity is highest after meat-diet and lowest after bread-diet. With reference to the amount of weight, the greatest quantity of gastric juice is required by meat, and the smallest quantity by milk (with reference to equivalents in nitrogen, more is needed by bread, and less by meat). Each variety of food calls forth every hour a definite amount of secretion and characteristic changes in the nature of the gastric juice. In case of bread-diet, the average amount of juice secreted every hour is one and a half times less than after milk or meat; the total amount secreted, however, is greater than in case of milk, because the digestion is prolonged over a longer period of time. A pause exists between the introduction of the food and the beginning of secretion, called the latent period, which lasts from four and a half to ten minutes. With reference to the work of the pancreas, it was found that in case of *"bread-juice"* amylolytic ferment mainly was excreted, and that in *"milk-juice"* the fat-splitting ferment predominated, and that the largest percentage of proteid-splitting ferment occurs in *"milk-juice,"* it being less in *meat- and bread-juice.* In prolonged nourishment with meat the proteolytic ferment increases progressively, while the amylolytic ferment gradually lessens in amount, and *vice versa* in case of a bread-diet.

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* J. P. Pawlow, The Work of The Digestive Glands, Translation, London and Philadelphia, 1902.

[24] Sticker, Wechselbeziehungen zwischen Speichel und Magensaft, Volkmann's Sammlungen klinischer Vorträge, No. 297.

[25] Biernacki, Die Bedeutung der Mundverdauung und des Mundspeichels für die Thätigkeit des gesunden und kranken Magens, Zeitschr. f. klin. Medicin, 1892, Bd. 21, Heft 1 u. 2.

[26] A. Schuld, Inaug.-Dissert., Leiden, 1892; Maly's Jahresb. für Thierchemie, 1893, S. 257.

If a dog in whom an œsophagotomy has been performed, is fed (sham-feeding), an abundant amount of gastric juice will appear at the gastric fistula in five minutes. There can be no doubt, therefore, but that nervous influence plays a rôle in this phenomenon. After the vagus has been severed (under proper experimental precautions), sham-feeding has no longer any influence upon the secretion of the gastric juice. It follows, therefore, *that the vagus is a secretory nerve to the gastric glands,* and hence it is possible to excite secretion by stimulation of that nerve. Secretion does not set in until after the lapse of a latent period (fifteen to sixty minutes), which may be explained by the probable simultaneous stimulation of inhibitory fibers. *The vagus is also the secretory nerve of the pancreas.* It also supplies that organ with inhibitory influences by the activity of inhibitory fibres, which directly affect its secretion. The sympathetic system also carries vasomotor constrictor as well as secretory fibres to the pancreas.

An abundant secretion of saliva is effected through psychical influence, as well as by the introduction of various substances into the mouth. It is of interest to know that in order to stimulate salivary secretion, the food must be in the dry state. A large quantity of gastric juice is secreted when *"sham-feeding"* is instituted, as well as when the animal is tempted with the sight of meat; this phenomenon is attributable to the eager longing for food and the feeling of contentment when the desire is satisfied (*"appetite-juice"*). On the other hand, chemical and mechanical stimulation of the oral and mucous membranes fails to excite reflexly the secretory nerves of the stomach.

The appetite is the main and strongest stimulant of the secretory nerves of the stomach. The psychic secretion is the normal commencement of the secretory activity of the stomach in the majority of cases. When dogs were fed with about 100 grammes of meat at intervals of one and a half hours, it was observed that the act of ingestion was accompanied every time by an increase in the digestive power and the quantity of the gastric juice. If meat be given *per os* to dogs which possess, besides the fistula of the small resected stomach, a second fistula of the larger stomach, it will soon pass out through the latter: the gastric juice will then be found to be identical, both in regard to the quality as well as quantity, with that of sham-feeding. No secretion follows the direct introduction of bread or boiled egg-albumen through the fistula into the larger stomach, although it does occur after the introduction of meat. The juice is, however, delayed in appearance (fifteen to forty-five minutes, instead of six to ten minutes); it is present in very minute quantities during the first hour (3 to 5 cubic centimetres, instead of 12 to 15 cubic centimetres), and possesses a very low digestive power. If about 100 grammes of meat are introduced directly into the stomachs of two dogs each, one of which is subjected to active sham-feeding, it will be found at the end of one and a half hours that the weight of the meat will have been reduced 30 grammes in the dog subjected to sham-feeding, and only 6 grammes in the other dog. The normal secretory curve represents a summation curve which is composed of the curve of the appetite-juice and that of the juice occurring after the direct introduction of meat. The stomach can be stimulated directly only specifically, and not mechanically. The glands may, however, become active by the mechanical action of the food upon the gastric walls, in combination with the effect of desire for food.

If food is introduced into the stomach of a dog by means of a tube, or, better, through a fistula, the following phenomena will be noted: Water is a chemic stimulant of gastric secretion, but only weakly so. The ash constituents of meat, as well as sodium chloride, soda, and hydrochloric acid, are wholly inefficient; indeed, soda may have an inhibitory action. Fluid egg-albumen, pure peptone, and the products of digestion which were obtained of the action of strong and pure gastric juice upon raw fibrine, do not excite secretion. Meat-broths, meat-juices, and solutions of meat-extracts are energetic stimulants of the gastric secretion. Starch, grape-sugar, and cane-sugar possess no influence. On the other hand, a combination of starch-paste and meat-extract doubles the quantity of the secretion. Fats do not stimulate secretion. After the stomach has once secreted juice which possesses the power to digest food, products are formed in the digestion of bread and egg foods which increase the digestive strength of the juice. The high digestive power claimed for "bread-juice" is truly remarkable, and is accounted for by the amalgamation of the albumen with the starch in the bread. Fat, when introduced into the stomach with other articles of food, limits secretion. When fat is directly introduced into the stomach it inhibits, both quantitatively and qualitatively, the juice secreted during "sham-feeding." The whole secretory activity of the stomach is therefore nervous and (excepting the psychical secretion) reflex in origin. Gastric secretion cannot be stimulated reflexly by way of the rectum, although such an effect may be obtained through the small intestines.

Hence we see that in the large majority of cases gastric digestion begins with an active central (automatic) stimulation of the secretory and trophic fibres of the glands of the stomach ("appetite-juice"). After an interval of more or less time following the ingestion of food, reflex stimulation begins in the stomach, while the automatic, psychical stimulation gradually disappears. When meat is introduced into the stomach, the centre for the secretory fibres is energetically stimulated (copious secretion) reflexly through the end-apparatus in the stomach; when bread is ingested, the trophic fibres are markedly stimulated through the end-apparatus (high digestive power).

We note, with reference to normal stimulation of the innervation apparatus of the pancreas, that acids possess the greatest power for stimulation of that organ. Hydrochloric, phosphoric, citric, lactic, and acetic acids are all equally effective in this respect, and when they are introduced into the stomach, pancreatic juice is secreted in from two to two and a half minutes. Pepper, mustard, etc., are ineffectual. Solutions of sugar, peptone, and albumen, when introduced into the stomach, do not affect the secretion of the pancreatic juice unless they possess an acid reaction; neutralisation of the latter causes inhibition of that secretion. Pancreatic secretion becomes active only when the acids pass from the stomach into the duodenum, from which point the pancreas is stimulated reflexly. If entrance into the duodenum is impossible, no pancreatic secretion can be excited by way of the stomach, nor is it possible to stimulate that gland by way of the rectum. Accordingly, the secretion of the pancreatic juice always follows the entrance of the acid chyme into the duodenum. The pancreatic juice, which flows upon the appearance of acid solutions in the duodenum, possesses a maximal percentage of inorganic constituents and only an insignificant amount of organic material. The pancreatic juice itself, and also its ash, are highly alkaline. Starch does not excite the secretion of pancreatic juice, but it causes an increase in the amylolytic ferment which that secretion contains. Fat is a true stimulant of pancreatic secretion, and increases its fat-splitting ferment. The secretion of the pancreatic juice does not cease during sleep. Although psychical stimulation of pancreatic secretion may exist, yet it plays a very unimportant part. Water is an independent stimulant of the pancreatic juice. The meat-extracts act only by means of the water they contain. Solutions of the alkalies, including both the salts and metals, possess an inhibitory influence upon the process; likewise solutions of sugar. The gastric juice of the fundus is characterised by a powerful mineral acid—*hydrochloric acid* —as well as three enzymes.

1. Hydrochloric Acid.

The percentage of hydrochloric acid in human gastric juice varies from 0.1 to 0.22 per cent., but in dogs it may reach 0.3 per cent. or even higher.*

Schmidt (1847) was the first to determine analytically the presence of hydrochloric acid in the gastric juice. After determining the total amount of bases present (potassium, sodium, calcium, magnesium, iron oxide, and ammonia) and the quantity of chlorine, it was found, after saturation of the bases with hydrochloric acid, that a certain amount of chlorine remained unaccounted for. This represented from 2.5 to 5 grammes of hydrochloric acid to a litre. The same result was obtained by means of titration with lime and baryta-water. The above-mentioned percentage of hydrochloric acid is not reached until at the height of digestion; at the commencement and end of digestion this percentage is markedly diminished.

Hydrochloric acid acts in six different ways, all of which are of great importance to the normal process of digestion.

1. Hydrochloric acid in the stomach is *antizymotic* and *antiseptic*, inasmuch as it prevents abnormal fermentation and destroys pathogenic organ-

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* Schoumow-Simanovski (Archiv für experimentelle Pathologie und Pharmakologie, 1894, Bd. 33) found an acid degree of 4.6 to 5.8 per mille in pure, unmixed gastric juice of dogs.

isms (Koch,[27] Falk,[28] Frank,[29] Wesener,[30] Miller,[31] Macfadyen,[32] Strauss and Wurtz,[33] Kurloff and Wagner,[34] Kast,[35] H. Hamburger,[36] and others) which enter the stomach with the ingesta or by other means. This antiseptic action is not limited to the stomach, for it continues effective in the duodenum and perhaps also in more distant sections of the intestines. A few investigators, amongst them B. Bunge,[37] have gone to the extreme of attributing to this antizymotic action of the hydrochloric acid the chief function of the stomach, which is, of course, obviously exaggerated.

2. Hydrochloric acid has the property of converting the inactive proenzymes of the stomach (pepsinogen, rennin-zymogen) into active enzymes in a short time (according to Langley, in one minute), and to large extent.

3. Hydrochloric acid is probably also of importance in regulating peristalsis. It appears, however, that in the absence of hydrochloric acid the same effect is obtained through the activity of regulatory centres.

4. Hydrochloric acid, in conjunction with pepsine, converts albuminoid bodies into peptones, gelatine into gelatine-peptones (peptone de colle), and elastine into elastine-peptones. Pepsine, is, however, the essential element in this process, for hydrochloric acid may be replaced to a certain extent by other acids (nitric acid, phosphoric acid, oxalic acid, sulphuric acid, lactic acid, butyric acid, etc.).

5. Hydrochloric acid converts cane-sugar into invert sugar (dextrose and lævulose). Hydrochloric acid shares this property with a number of bacteria, which likewise invert cane-sugar, although they require a longer period of time.

6. Hydrochloric acid is the chief stimulant of pancreatic secretion, as was proven by the experiments of Dolinsky[38] and Walter[39] (under Pawlow) (see page 25). The regulation of the pancreatic secretion depends upon the acidity of the gastric contents and upon their passage into the duodenum

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[27] Koch, Mittheilungen aus dem Kaiserlichen Gesundheitsamt, 1881 and 1884.

[28] Falk, Virchow's Archiv, 1883, Bd. 93.

[29] Frank, Deutsche medicinische Wochenschrift, 1884, No. 20.

[30] Wesener, Fütterungstuberkulose, Freiburg, 1885.

[31] Miller, Deutsche medicinische Wochenschrift, 1885, No. 49; 1886, No. 8.

[32] Macfadyen, Journal of Anatomy and Physiology, 1887, Bd. 21.

[33] Strauss et Wurtz, Archive de Médecine expérimentale, 1889, Bd. 1, No. 3.

[34] Kurloff and Wagner, Wratsch, 1889, No. 42 and 43.

[35] Kast, Festschrift zur Eröffnung des neuen Krankenhauses zu Hamburg-Eppendorf, 1889.

[36] H. Hamburger, Centralblatt für klinische Medicin, 1890, No. 42.

[37] Bunge, Lehrbuch der physiologischen und pathologischen Chemie, Leipzig, 1887.

[38] Dolinsky, Inaug.-Diss., Petersburg, 1894; Ref. nach Malay's Jahresbericht, Bd. 24, resp. 29.

[39] Walter, Inaug.-Diss., Petersburg, 1897; Ref. nach Maly's Jahresbericht, Bd. 24, resp. 29.

at regular intervals of time. It is furthermore worthy of note that secretion of the pancreas is brought about reflexly only by uncombined hydrochloric acid. Hence it is probable that the varying affinity of foodstuffs to hydrochloric acid is a factor of great influence upon the course and quantity of pancreatic secretion.

Theories of Hydrochloric Acid Secretion.

Many theories have been offered in explanation of the secretion of hydrochloric acid. The hypothesis of Maly is the most acceptable. According to that author, the blood is a fluid, alkaline in reaction, which is, however, derived from the presence of two acid salts, bicarbonate of sodium ($NaHCO_3$) and disodium phosphate (Na_2HPO_4). The blood contains an excess of carbonic acid, which, acting upon the disodium phosphate, produces monosodium phosphate, according to the following equation:—

$$Na_2HPO_4 + CO_2 + H_2O = NaHCO_3 + NaH_2PO_4$$

The presence of sodium chloride (or calcium chloride) causes hydrochloric acid to be set free, as follows:—

$$NaH_2PO_4 + NaCl = Na_2HPO_4 + HCl$$

This acid, however, possesses a higher diffusive power than any other acid, and it is then only necessary to assume, in order to account for the separation of hydrochloric acid, that the gastric glands act as diffusion-apparatus. Many objections have been raised against this theory offered by Maly, but too much space would be required to discuss them here.

More recently a different theory was promulgated by H. Köppe,[40] in which he attributes the formation of hydrochloric acid to the gastric contents themselves, that is, the chlorides they contain. Sodium chloride in the stomach is, namely, separated into sodium and chlorine ions; the walls of the stomach are permeable to the former, but not to the latter. Hydrogen ions, due to dissociation of the water, pass from the blood into the stomach. Accordingly, the stomach contains chlorine ions and hydrogen ions, the constituents of free hydrochloric acid. This theory has also met with many objections, foremost of which are the above experiments of Pawlow, who showed that a blind gastric pouch, opening externally, and which is free of food, secretes hydrochloric acid reflexly. Köppe's theory is furthermore contradicted by the investigations of Wesener.[41] The latter demonstrated that a fasting stomach, which has been washed out and is free of chlorine, may show the presence of hydrochloric acid secretion, when stimulated mechanically with Turck's gyromele; this does not occur when a physiologic normal salt solution is introduced. We are therefore compelled, for the present, to look upon the secretion of hydrochloric acid as a specific function of the gastric glands.

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[40] H. Köppe, Pflüger's Archiv, 1896, Bd. 62, S. 567.

[41] Wesener, Pflüger's Archiv, 1899, Bd. 77, S. 483.

2. Pepsinogen and Pepsine.

Pepsine converts albuminoid bodies, in the presence of free hydrochloric acid, into peptones, and deprives gelatine of its property of forming a jelly, converting it into gelatine-peptone. No other mineral acid, still less the organic acids, gives in combination with pepsine similar or better digestive results than pepsine-hydrochloric acid. This seems to be due to the fact that hydrochloric acid is able to convert pepsinogen into pepsine more rapidly and more thoroughly. According to the investigations of Langley and Edkins,[42] as well as of Podwyssotzki[43] and Herzen,[44] the secretion of the glands of the fundus consists only in the production of the inactive proenzyme.

This is characterised not only by its rapid conversion into pepsine by means of hydrochloric acid, but also by its relationship to sodium carbonate and carbonic acid. The former destroys pepsine within a few minutes, and the latter destroys pepsinogen quickly, while pepsine is extinguished by it more slowly. In general, pepsinogen possesses a high power of resistance and is rarely completely destroyed, even in marked changes in the gastric mucosa, in catarrh, and even in gastric cancer.

The peptones, which are the products of the activity of pepsine and hydrochloric acid, present certain important practical properties. The conversion of albuminous bodies and gelatine substances proceeds, as in case of starch, not at once, but in stages. It is a difficult matter to determine exactly the intermediate products, since we have to deal with a "series in motion," during which their composition fluctuates every moment. One may, however, isolate several fairly definite chemical combinations from the complicated albuminous mixture, from the presence of which (see examination of gastric contents) certain conclusions may be drawn. The first combination which arises is *acid-albumen,* also called syntonin. It represents simply a more or less close union between albumen and acid. Syntonin has the property of remaining in solution in the presence of acids and alkalies, but it is precipitated in a neutral reaction (therefore it is also called neutralisation precipitate). Continuation of activity of the gastric juice produces next the formation, principally, of *albumoses,* while true peptones, as understood by Kühne, (so-called amphopeptones) are produced only in minute quantities.

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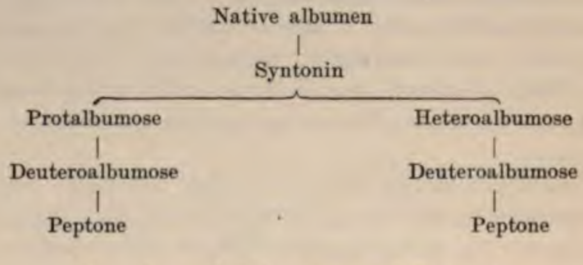
[42] **Langley and Edkins,** Journal of Physiology, 1886, Bd. 7, S. 371-415.

[43] **Podwyssotzki,** Pflüger's Archiv, 1886, Bd. 39, S. 62-74.

[44] **Herzen, Annali** di chim. e di farmac., Bd. 8, S. 302; Ref. nach Maly's **Jahresberichte für** Thierchemie, 1888, Bd. 18, S. 193.

The more recent investigations of Kühne and Chittenden[45], Wenz[46], R. Neumeister,[47] and others have considerably changed our earlier views on the gradual conversion of albumen under the influence of the gastric juice. According to their findings, two different albumoses (hitherto designated as propeptone or hemialbumose) arise from syntonin, namely, protalbumose and heteroalbumose.

Each of these albumoses is converted into a deuteroalbumose, the properties of which, however, differ from each other only slightly. Not until these deuteroalbumoses undergo further conversion true peptone is formed. In order to make these relationships clearer the following schema is offered:



Reactions of Albumoses and Peptones.

The term *"albumoses"* was formerly employed to designate albuminous bodies which do not coagulate on boiling. Of these, special attention was paid to hemialbumose, which is precipitated in a cold acetic acid solution by saturation with sodium chloride, and which is dissolved by heat. The same reaction followed also the superaddition of nitric acid. Hemialbumose was also precipitated from a solution by the addition of acetic acid and potassium ferrocyanide. The term *"peptones"* was formerly employed to indicate such albuminous bodies as were easily soluble in water and not coagulable by heat, and which, contrary to hemialbumoses, were precipitated from their solutions neither by nitric acid, nor by neutral salts and acids, nor by acetic acid and potassium ferrocyanide. The following characteristics were formerly ascribed to peptones and albumoses: they yield all the colour-reactions of albumen, especially the biuret test, which, however, contrary to albumen, give a purple shade. They are precipitated by ammoniacal solution of the subacetate of lead, perchloride of mercury, alcohol, tannic acid, phosphotungstic and phospho-molybdic acids, hydrargyro-iodide of potassium, and hydrochloric acid, and finally picric acid. Albumoses and peptones are furthermore more diffusible than the native albumens, a property which increases with the amount of peptone contained.

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[45] Kühne and Chittenden, Zeitschrift für Biologie, 1883, N. F. Bd. 1, S. 159; *ibid.*, 1884, Bd. 2, S. 11; *ibid.*, 1886, Bd. 4, S. 423. W. Kühne, Verhandlungen des naturhistorischen Vereins zu Heidelberg, 1885, N. F. Bd. 3, S. 286; Zeitschrift für Biologie, 1893, N. F. Bd. 11, S. 1.

[46] Wenz, Zeitschrift für Biologie, 1886, N. F. Bd. 4, S. 11.

[47] R. Neumeister, Zeitschrift für Biologie, 1887, N. F. 5, S. 381; furthermore, 1888, Bd. 6, S. 267. Compare also this author's text-book on physiologic chemistry, 1893, S. 182 *et seq.*

Recently the experiences of Heynsius and Kühne have led to the employment of ammonium sulphate* as a means of separating albumoses from peptones.

The products of digestion which are precipitated by ammonium sulphate in substance are termed albumoses, whereas those which become dissolved are, on the other hand, true peptones. With reference to the reactions of the individual albumoses, it may be stated, as a rule, that the more advanced the digestion of albuminous bodies, the more difficult is the precipitation of albumoses by means of chemical agents.

Individually, the various products of digestion are distinguished, according to Neumeister, by the following reactions:—

The *primary* albumoses may be precipitated from neutral solutions by means of sodium chloride, whereas a neutral solution of *deuteroalbumoses* remains clear, and a precipitation ensues only upon the addition of an acid. Nitric acid precipitates protoalbumose and heteroalbumose without the presence of salts; the deuteroalbumoses are, however, not at all precipitated thus, or only in the presence of salts, and then only to a slight extent. Potassium ferrocyanide and acetic acid, an excess of picric acid, as well as a neutral solution of copper sulphate, precipitate the primary albumoses with only a little more difficulty than the albumens; the deuteroalbumoses are, on the other hand, precipitated by acetic acid and potassium ferrocyanide only after long standing, and pure deuteroalbumose is not at all precipitated by copper sulphate.

The true peptones form honey-coloured amorphous powders, which are extraordinarily hygroscopical and possess a nauseating and bitter taste. The addition of a little water causes them to sizzle loudly, like anhydrous phosphoric acid, with evolution of considerable heat. Peptones are distinguished from albumoses by the following reactions: they are wholly indifferent to saturation with neutral salts and are more easily diffusible than the albumoses. The majority of the precipitating agents of the albuminous bodies and the albumoses (nitric acid with or without common salt, acetic acid and potassium ferrocyanide, excess of picric acid, trichloracetic acid, hydrargyro-iodide of potassium and hydrochloric acid) remain ineffectual in case of peptones. These are only precipitated by absolute alcohol, tannic acid, phosphotungstic acid, and perchloride of mercury.

The above-mentioned reactions refer only to the albumoses of fibrine, although other albuminous bodies show analogous results.

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* According to Römer and E. Zunz, zinc sulphate may also be used for this purpose.

Kühne's schema of the formation of albumoses has recently met with much opposition (Hamburger, Morochowetz, Umber, Alexander, E. Zunz, and others). The elementary form of deuteroalbumoses has been strongly disputed.

Hydrochloric acid and pepsine must bear a definite relationship to each other as regards the amount present, *i.e.,* the acid must not become excessively high in proportion to the quantity of the pepsine. The right amount of the acid equals 0.25 per cent. (according to the investigation on natural gastric juice by Dr. Trzebinski in my laboratory); higher and lower percentages of hydrochloric acid retard albuminoid digestion.

Quantitative Estimation of Pepsine.

Various methods may be resorted to for the quantitative estimation of pepsine. Those suggested by Brücke, Grützner, Mett, and Schütz are recommended as most useful for physiologic purposes.

1. *Method of Brücke.*[48] A sufficient amount of hydrochloric acid is added to each solution to be compared, until 1 per mille of that acid is present. At the same time an aqueous solution of hydrochloric acid of the same strength is prepared. The first digestive mixture to be tested is then diluted one-half, one-third, one-fourth, one-eighth, etc., with the 1 per mille aqueous solution of hydrochloric acid. The second digestive mixture is treated in the same manner. A flocculus of fibrin is now placed in each of the diluted fluids. By comparing the digestion of the fibrine in the different solutions, useful figures may be obtained, which will express the *relative* amount of pepsine present. If, for example, we designate the solutions to be compared as *A* and *B,* and find that one-half of *A* corresponds to the digestive fluid *B,* it may be concluded that *A* contains twice as much pepsine as *B.*

2. *Method of Grützner.*[49] This is a calorimetric method, and depends upon the comparative estimation of the colour-intensity of carmine fibrine,* after being acted upon by solutions of pepsine. For purposes of comparison a scale of colours, consisting of ten grades, is employed. These consist of solutions of carmine in the proportion of 19.9 of water to 0.1 glycerine-carmine, 19.8 water, and 0.2 glycerine carmine, etc.

3. *Method of Mett.*[50] Egg-albumen is drawn into a narrow glass tube (diameter of 1 to 2 millimetres) and coagulated at a temperature of 95° C. After cooling, the glass tube is cut into small sections (about 3 to 5 centimetres long). Two of these are placed in each glass beaker containing the

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* Carmine fibrine is prepared, according to Grützner, by placing fibrine for twenty hours in an ammoniacal solution of carmine. It is then washed and placed in a dilute solution of hydrochloric acid until it becomes swollen.

[48] Brücke, Sitzungsberichte der Wiener Akademie, Bd. 37, S. 131.

[49] Grützner, Archiv für die gesammte Physiologie, 1874, Bd. 8, S. 452.

[50] Samojloff, Archive des sciences biologiques, Bd. 2, S. 698-729.

acid-and-pepsine solution. The beaker is then set in a thermostat at a temperature of 37 to 40° C. for ten hours. A millimetre rule is then placed parallel to the small tubes, and the length of the partially-digested columns of egg-albumen is measured with the aid of a low-power microscope. The measurement obtained by subtracting the length of the partially digested columns of egg-albumen from the original length, expressed in millimetres, represents the peptonising strength of the digestive fluids.

4. As has been shown by E. Schütz,[51] and confirmed by Borissow[52] and Huppert,[53] the relative amount of pepsine may, for physiological purposes, be estimated from the quantity of resulting albumoses. The resulting products of peptic digestion are proportionate in amount, all other things being equal, and within certain limits, to the square root of the relative amount of pepsine. (Schütz-Borissow's law.) Thus, for example, if in two comparisons, 2 millimetres of egg-albumen were dissolved in the one test, and 3 millimetres in the other, the amount of pepsine would represent the proportion of 4 : 9. Beyond a certain degree of concentration this law does not hold true. See Chapter VI for the quantitative estimation of pepsine in the gastric contents for diagnostic purposes.

3. Rennin-Zymogen and Rennin-Ferment.

(Lab-zymogen and Lab-ferment.)

The normal human stomach contains, even from the earliest childhood on (Leo), an enzyme which has the property of coagulating milk in neutral solution, *i.e.*, to precipitate its caseine. Credit is due to the famous Swedish physiologist, Hammarsten[54] (1872), for having discovered and studied in greatest detail the rennin-ferment. It was studied in man first by Schumburg,[55] and then by myself,[56] Raudnitz,[57] (in sucklings), Klemperer,[58] Johnson,[59] C. Rosenthal,[60] Leo,[61] Arthus and Pagès,[62] and others, in the healthy and sick. My investigations in healthy persons, which were confirmed in all essential points by C. Rosenthal, showed that the rennin-ferment appears simultaneously with hydrochloric acid, and then increases parallel to the increase of that acid, and lessens in amount as the acid diminishes. Like pepsine, lab-ferment also possesses a previous stage known as *lab-zymogen,* which, when treated with acids, is rapidly converted into lab-enzyme; hydrochloric acid and sulphuric acid act most rapidly, then follows nitric acid, and next oxalic and phosphoric acids. Lactic acid is much weaker in action, while acetic acid is the weakest (Lörcher[63]). Rennin-enzyme shares, furthermore, with pepsine the peculiarity of being destroyed by alkalies even in weak concentration, whereas rennin-zymogen, like pepsinogen, remains practically unaffected by them. On the other hand, according to my investigations (*l.c.*) rennin-enzyme is not at all affected by pepsine, saliva, or fat, and only by bile in so far that it deprives the gastric juice of a portion of its acid and thus interferes in the conversion of pro-enzymes into active enzymes. Rennin may be totally destroyed in a strongly active infusion of pure gastric mucous membrane mixed with 3 per cent. hydrochloric acid, by heating it to a temperature of 37 to 40° C. for forty-eight hours, whereas pepsine remains intact. To what extent this fact will hold true for human gastric contents has not yet been determined.

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[51] E. Schütz, Zeitschr. f. physiol. Chemie, 1885, Bd. 9, S. 577.

[52] Borissow, Inaug.-Diss., Petersburg, 1891.

[53] E. Schütz and Huppert, Pflüger's Archiv, 1900, Bd. 80, S. 470-526.

[54] Hammarsten, Upsala Laekareförenings Förhandlinger, 1872, Bd. 2.

[55] Schumburg, Virchow's Archiv, 1884, Bd. 97, S. 260.

[56] Boas, Centralblatt f. d. med. Wissenschaften, 1887, No. 23; Zeitschr. f. klin. Med., 1888, Bd. 14, S. 249-279.

[57] Raudnitz, Prager med. Wochenschrift, 1887, No. 24.

[58] Klemperer, Zeitschr. f. klin. Med., 1888, Bd. 14, S. 280.

[59] Johnson, *ibid*, S. 240.

[60] C. Rosenthal, Berliner klin. Wochenschrift, 1888, No. 45.

[61] Leo, Berliner klin. Wochenschrift, 1888, No. 49.

[62] Arthus and Pagès, Comptes rendus hebdomadaires des séances de la société de Biologie, 1891, 13 Fév., S. 131.

The test for the rennin-ferment is made in the following simple manner: equal amounts of exactly neutralised gastric juice or gastric contents, and boiled or unboiled milk,* are mixed (raw milk coagulates more rapidly than boiled milk) and placed in an incubator (37° to 40° C.) The presence of the rennin-ferment is demonstrated by the appearance, in a short time (ten to fifteen minutes), without a change in the reaction of the mixture, of fine flocculent caseine curds, which gradually thicken into a caseine clot.

Rennin-zymogen is tested for by slightly alkalinising** the gastric contents with dilute potash or soda solution (whereby the rennin-enzyme is destroyed, but the zymogen remains unaffected), and then adding 2 to 3 cubic centimetres of a 1-per-cent. solution of calcium chloride. If rennin-zymogen is present, caseine curds develop shortly in a like manner as in case of the rennin-enzyme. Lörcher (*l.c.*), though, states that the alleged zymogen action is nothing else but rennin-enzyme which has not been thoroughly destroyed by the alkali.

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* I claim, contrary to Leo (*l.c.*), that the employment of boiled milk for the detection of rennin-ferment is preferable to raw milk, because primarily all bacterial action is prevented, and secondarily because the more gradual coagulation furnishes better results as to an increase or decrease in rennin.

** An increased amount of hydrate of potassium solution also destroys the rennin-zymogen.

[63] Lörcher, Pflüger's Archiv, 1897, Bd. 69, S. 141.

We may, however, safely exclude this possibility, at least in regard to human rennin-zymogen, in view of the fact that I have proven in my investigations that the true ferment was never present simultaneously with the zymogen.

Acording to Ivar Bang[64] the rennin of a pig's stomach is not *chymosin*, as was formerly supposed, but a varied form of it, which he called *parachymosin*. The mucous membrane of the stomach of a calf, as well as of fish, contains, on the contrary, chymosin. The rennin-ferment of man is also supposed to be derived from parachymosin, although the investigations on this subject are not yet completed.

The essential differences between parachymosin and chymosin are, according to Bang, as follows: Chymosin, when treated with 0.2 to 0.4 per cent. hydrochloric acid, is completely destroyed in twenty-four to forty-eight hours; whereas parachymosin, on the contrary, after neutralisation with alkali, remains active after an elapse of even twice the above time; and if, as digestion continues, the action on milk ceases after neutralisation with alkalies, it may again become active by neutralisation with carbonate of lime ($CaCo_3$). This late action, which ceases only after long-continued digestion, is due to the fact that calcium chloride has a very much more accelerating influence upon milk-coagulation by parachymosin than upon that by chymosin. In the coagulation of milk by chymosin, the dilution of the enzyme solution with water acts in such a manner that the time of coagulation is indirectly proportionate to the amount of ferment; but when a solution of parachymosin is diluted with water, the time of coagulation is decidedly less. A solution of parachymosin is, in the presence of a proportionate amount of hydrochloric acid (about 0.1), much more resistent when heated to 70°, than a solution of chymosin under similar circumstances. On the other hand, a solution of parachymosin is much more sensitive to alkalies than a solution of chymosin.

Quantitative Estimation of Rennin.

As has been demonstrated by the experiments undertaken by myself and Dr. Trzebinski, the amount of rennin-ferment and rennin-zymogen in the gastric contents may be fairly accurately estimated, as in case of pepsine by means of gradual dilution. For rennin-ferment, the extreme limit of dilution should be 1 : 30 or 40; for rennin-zymogen, 1 : 75 to 1 : 150. This method furnishes important diagnostic conclusions as to the secreting power of the gastric glands, and will be considered in greater detail in the section on the examination of the gastric contents (Chapter VI).

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[64] Ivar Bang. Deutsche medicinische Wochenschrift, 1899, No. 3, u. Pflüger's Archiv, 1900, Bd. 79, S. 425.

4. The Fat-splitting Ferment.

Cash[65] and Ogata[66] quite some time ago made the observation that in animals traces of fatty acids are formed from neutral fat or oil in the stomach. Later this theory was confirmed by Klemperer and Scheuerlen,[67] who demonstrated that in a doubly-ligated stomach of a dog 1 to 2 per cent. of fatty acids may be derived from neutral oil. Only in case of marked fermentation does the splitting of fats equal 6 per cent., according to the above authors. F. Volhard[68] is credited with having shown that the normal gastric mucous membrane in man, as well as in animals, produces a *fairly characteristic and marked fat-splitting ferment.* This ferment, similar to steapsin in the pancreatic juice, acts most readily upon emulsified fats as they appear in nature in the yolk of egg, milk, and cream. The splitting of fat is effected to a high degree (to 70 per cent.). The ferment may be extracted by means of glycerine, but only from the fundus of the stomach, for the pyloric end furnishes no fat-splitting ferment. Volhard was able, in a certain series of experiments, to apply the Schütz-Borissow law also (page 33) to the fat-splitting enzyme, although he obtained contradictory results in other tests. Of considerable interest, furthermore, is the fact that, like pepsine and rennin-ferment, the fat-splitting ferment is also destroyed by traces of alkali. On the other hand, the glycerine extract of the fat-ferment can endure a noteworthy amount of caustic alkali without influence upon its activity. Then, again, hydrochloric acid lessens the fat-splitting action of the glycerine-extract, whereas that of the gastric juice remains unaffected by the acid in the same strength. We must assume, therefore, that, as in the case of pepsine and rennin-ferment, a zymogen exists for the fat-splitting ferment. The hydrochloric acid of the stomach is not necessary for the splitting of fat, for neutralised gastric juices act as favourably as the original.

At body temperature both rapidly lose their action, the neutralised solution more rapidly than the acid; the glycerine extract, on the contrary, is not affected. Boiling and alcohol destroy this ferment and its zymogen. Refer to the Chapter on Examination of the Gastric Contents for the action of the fat-splitting ferment under pathologic conditions.

In recent years the question has been frequently asked whether the stomach possesses the property of excreting substances foreign to the body. After this question had been answered positively by a large number of investigators (Leipeweber,

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[65] Cash, du Bois Archiv, 1880; Physiol. Abth., S. 323.

[66] Ogata, *ibid*, 1881, S. 515.

[67] Klemperer and Scheuerlen, Zeitschrift f. klin. Medicin, 1889, Bd. 15, S. 370.

[68] F. Volhard, Münchener medicin. Wochenschrift, 1900, No. 5-6; Zeitschr. f. klin. Medicin, 1901, Bd. 42; Verhandl. des 19. Kongresses für innere Medicin in Berlin, 1901.

Blanchier and Rochefontaine, Kandidoff, and Bongers), Nencki proved, and I believe without meeting opposition, that such an excretion does not occur. Nencki obtained a result which coincided with the claims of the above authors only when the gastric juice was mixed with bile.

Fermentation in the Normal Stomach.

Since we introduce with the food into our stomachs various micro-organisms, which cause fermentation, it is easy to conceive of their multiplication under favourable circumstances, and thus causing further fermentation. On the other hand, the fine investigations of Miller[69] have demonstrated the presence in the saliva or the oral cavity of micro-organisms (lactic acid bacilli and others) capable of producing fermentation, which, when introduced into the stomach, cause fermentative changes. This is *a priori* the more probable because, according to Miller's researches, fermentation does not become inhibited until the degree of acidity is as high as at the height of digestion. This refers particularly to carbohydrate fermentation, for fermentation of the proteids does not occur at all in an acid reaction, or only to a very limited degree. Before discussing the fermentation of the carbohydrates themselves, it is necessary to refer to the above-mentioned forms of fermentative changes.

I. *Lactic Acid Fermentation.* In this process of fermentation the sugar of milk ($C_{12}H_{22}O_{11}$) is simply split into four molecules of lactic acid $= 4\ (C_3H_6O_3)$, which occurs probably after the former has previously undergone conversion into two molecules of glucose $= 2\ (C_6H_{12}O_6)$. In other cases, aside from the lactic acid, we may note the occurrence of carbonic acid, hydrogen, as well as acetic, formic, succinic, and perhaps also butyric acids.

Pasteur[70] demonstrated as early as 1857 that the formation of lactic acid from sugar of milk depended upon the presence of a micro-organism (ferment lactique). Hueppe[71] was, however, the first to cultivate a distinct bacillus from milk by Koch's method (Fig. 7).

This micro-organism, which Hueppe designated as the *bacillus acidi lactici*, consists of short, plump rods 1 to 1.7 μ in length and 0.3 to 0.4 μ in thickness. It is immobile and produces spores. The temperature limits favouring its development are between 10° and 45.5° C., the optimum being between 35° and 42° C. Aside from the production of lactic acid, this bacillus possesses properties of inversion, and is furthermore capable of splitting also cane-sugar, sugar of milk, and mannite into lactic acid, with the simultaneous development of carbonic acid. Aside from the bacillus of lactic acid, a number of other micro-organisms (about fifteen) exist which produce lactic acid in carbohydrate solutions. Miller[72] cultivated a fungus from the saliva, which seems to be identical, both morphologically and physiologically, with the organism discovered by Hueppe. Whether it also causes lactic acid fermentation in the stomach, or whether it does so only in combination with several varieties of bacteria, which is more probable, has not yet been determined.

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[69] Miller, Deutsche medicinische Wochenschrift, 1885, No. 49.

[70] Pasteur, Annales de Chimie et de Physiologie, 1857.

[71] Hueppe, Mittheil. a. d. Kaiserl. Gesundheitsamt, Bd. 2, S. 307.

Lactic acid fermentation ceases in the presence of hydrochloric acid, even in very small amounts (over 0.7 per mille), according to the researches of F. Cohn,[73] E. Hirschfeld,[74] H. Strauss and Bialocour,[75] all of whom agreed upon this point.

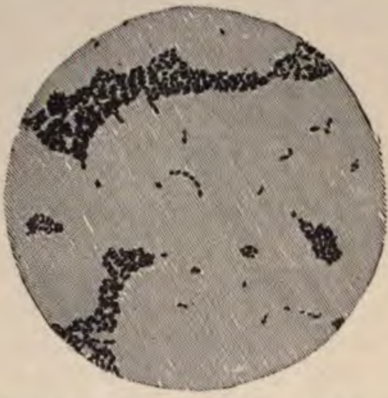

Fig. 7.—Lactic Acid Bacillus (Hueppe). (After C. Günther.)

II. *Butyric Acid Fermentation.* The investigations of Fitz[76] and Flügge[77] tend to prove that the development of butyric acid fermentation is due to various forms of bacteria. The majority of micro-organisms causing butyric acid fermentation are anaërobic, on account of which great difficulties are met in obtaining pure cultures. The best-known of these is the fungus of butyric acid fermentation studied by Prazmowski[78] (*bacillus butyricus* or *clostridium butyricum*). It consists of a rod-shaped bacillus, 2 to 12 μ long, and grows either singly, or in long chains, or in the form of zoöglœa. The formation of spores causes peculiar changes, which lead to the development of spindle-, ellipsoid-, and tadpole-shaped varieties (Fig. 8). The entrance of air stops or destroys the fermentative action of the bacillus butyricus.

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[72] Miller, Die Mikroorganismen der Mundhöhle, Leipzig, 1892, 2 Aufl.

[73] F. Cohn, Zeitschr. f. physiol. Chemie, 1890, Bd. 14, S. 75.

[74] E. Hirschfeld, Pflüger's Archiv, 1890, Bd. 47, S. 510.

[75] Strauss u. Bialocour, Zeitschr. f. klin. Medicin, 1895, Bd. 28, S. 567-578.

[76] Fitz, Ber. d. deutsch. chem. Gesellschaft, Bd. 15, S. 867-881.

[77] Flügge, Die Mikroorganismen, Leipzig, 1886, S. 296.

[78] Prazmowski, Untersuchungen über die Entwicklung und Fermentwirkung einiger Bakterienarten, Leipzig, 1880.

The bacillus butyricus reacts characteristically with iodine, which, together with its shape, facilitates the detection of the fungus in mixtures. Iodine colours it blue to dark violet, a colour reaction which occurs, however, only in starch-cellulose solutions and in the presence of glycerine and calcium lactate, whereas it is generally absent in solutions of dextrine and sugar. I have observed the characteristic butyric acid bacillus several times in the secretion of a fasting stomach; and, as is well known, it may be found in large numbers in the fæces under normal conditions; they are seen to lie both singly and in large groups in the shape of a lemon or a whetstone. I cannot state whether the presence of the bacillus butyricus under such circumstances should be regarded as pathological. Butyric acid fermentation occurs in milk, according to Flügge,[79] after lactic acid has been previously produced. The process may be demonstrated as follows:—

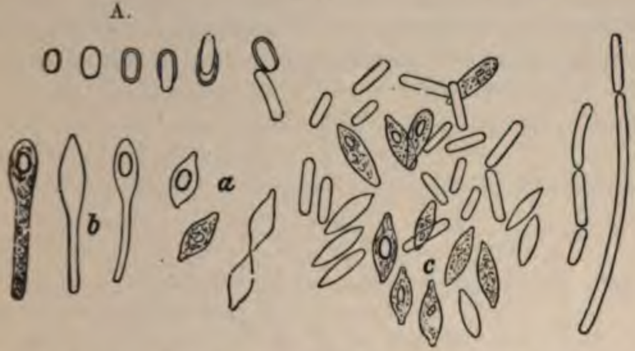

Fig. 8.—Bacillus Butyricus. *a*, *b*, Tadpole- and spindle-shaped forms, partly with spores. *c*, Zoöglœa form. *A*, Germination of a spore. (After Prazmowski.)

$$\underset{\text{Lactic acid}}{2\,(C_2H_6O_3)} = \underset{\text{Butyric acid}}{C_4H_8O_2} + \underset{\text{Carbonic acid}}{2CO_2} + \underset{\text{Hydrogen}}{4H}.$$

Hence we note that, aside from the production of butyric acid, carbonic acid and hydrogen are also set free. Lactic acid fermentation creates, moreover, conditions favourable to the production of butyric acid, in so far as the bacillus of lactic acid absorbs the oxygen.

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[79] Flügge, *l. c.*

III. *Acetic Acid Fermentation.* Acetic acid fermentation may occur under various circumstances. In one case it may follow the action of the fungus of acetic acid (mycoderma aceti) upon alcohol, with development, at first, of aldehyde, which, in turn undergoing oxydation, forms acetic acid (Fig. 9).

$$C_2H_6O + O = C_2H_4O + H_2O$$

Alcohol Aldehyde

$$C_2H_4O + O = C_2H_4O_2$$

Aldehyde Acetic acid

It is claimed that the same fungus causes the formation of small quantities of CO_2 and H_2O (Naegeli). This conversion does not occur in the stomach, because acetic acid fermentation ceases already at a temperature of 35° C.; also because the movements of the stomach produce conditions unfavourable to the development of the mycoderma aceti, which grows only on the surfaces. Finally, according to the experiments of F. Cohn,[80] the development of acetic acid fermentation is prevented by the presence of hydrochloric acid in so small amounts as 0.05 per mille.

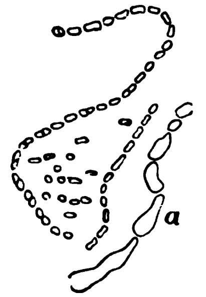

Fig. 9.—The Fungus of Acetic Acid Fermentation. *a*, Involution forms. 700:1.

IV. *Yeast Fermentation.* This depends upon the presence of various forms of saccharomyces (saccharomyces cerevisiæ, vini), all of which possess the property of splitting up glucose with the formation of alcohol and carbonic acid, according to the following formula:—

$$C_6H_{12}O_6 = 2\ (C_2H_6O) + 2CO_2$$

Glucose Ethylalcohol Carbonic acid

This process depends, according to Buchner, upon the formation of a specific ferment (*zymase*), which may also be isolated from yeast. Cane-sugar and sugar of milk are also fermented, but only after previous conversion into glucose; the cane-sugar being converted into dextrose and lævulose, and the sugar of milk (by means of the ferment of certain fungi found in the air) into galactose and dextrose. As has already been shown by Pasteur, the fermentation of glucose is, however, accompanied simultaneously by the production of glycerine and succinic acid and traces of acetic acid and amylalcohol. It is, however, not determined whether the action of other organisms can be excluded absolutely.

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[80] F. Cohn, *l. c.*

The optimum of the activity of yeast is 25° C., although the temperature-range varies according to certain circumstances. The action of yeast is hindered by free alkalies, sulphurous acid, perchloride of mercury, and chloroform, whereas sulphuretted hydrogen, arsenious acids, carbolic acid, strychnine, and hydrocyanic acid do not interfere with its activity at all, or only when present in a rather high degree of concentration. As we have repeatedly experienced, hydrochloric acid, in the strength found normally in the stomach, has no effect upon this process.

V. *H_2S Fermentation.* Refer to the Chapter on Examination of the Gastric Contents.

The following three possibilities must be distinguished in discussing lactic acid fermentation in the normal stomach:—

1. The presence of lactic acid, due to the introduction of lactic acid bacilli, which may be capable of propagation, in favourable nutritive media (for example, in sour milk, buttermilk, sauerkraut, pickles, etc.).

2. The presence of lactic acid, due to the introduction of lactic acid, through lactic acid producers which are no longer reproductive (all forms of bread, beer, etc.).

3. Presence of lactic acid in the stomach when carbohydrates free of lactic acid are introduced (for example, amylum).

In the first case the lactic acid fermentation may continue under favourable circumstances also in the normal stomach, yet the results of investigations are still uncertain on this point.

In the second case, under normal conditions, it would be expected to find only an amount of lactic acid corresponding to that introduced. Finally, when simple (free of lactic acid) carbohydrates are introduced, the researches of Martius and Lüttke[31] and myself,[32] show that not any, or only traces of, lactic acid are produced. The production of lactic acid in the stomach to any larger extent should therefore be regarded as pathological (for further information, refer to the Chapter on Examination of the Gastric Contents).

Aside from the above-mentioned forms of fermentation, we note in the stomach the "mucus or mannit fermentation," to which Frerichs has already called attention. It leads to the production of viscose, mannite, and carbonic acid. Black has observed a similar fermentation due to the activity of various fungi of the mouth in solutions of sugar. To what extent this may occur physiologically—Frerichs found it only in vomited material—must be left undecided.

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[31] Martius and Lüttke, Die Magensäure des Menschen, Stuttgart, 1892.

[32] Boas, Deutsche medicinische Wochenschrift, 1893, No. 34; Münchener med. Wochenschr., 1893, No. 43.

As has been proven by Tappeiner,[83] fermentation of *cellulose* occurs in the intestinal tract (of ruminants). The micro-organisms causing fermentation have not as yet been obtained in pure culture. Tappeiner claims that two varieties of cellulose fermentation exist; in the one type, carbonic acid and methan escape, together with small quantities of H_2S, aldehyde, isobutyric and acetic acids. In the second form of fermentation, carbonic acid and hydrogen alone arise, together with the same by-products as in the first case.

Absorption in the Stomach.

The process of absorption in the stomach has hitherto only been studied in an exact manner in animals. v. Anrep,[84] Smith,[85] and Tappeiner[86] ligated the pylorus of fasting animals and introduced peptones, sugar solutions, or diluted alcohol into the stomach. The animals were killed later and the gastric contents examined. Another experiment consisted in establishing a gastric fistula in the neighbourhood of the pylorus, through which a rubber bag capable of being filled with water was introduced to close the pyloric opening; substances suitable for absorption were then introduced into the stomach. v. Anrep and Smith concluded from these experiments that the stomach absorbed sugar and peptones, whereas Tappeiner found the stomach only slightly adapted to this function, but that it was suited for the absorption of dilute alcohol. More recently the absorption processes were carefully determined by v. Mering,[87] Moritz,[88] Hirsch,[89] and Brandl.[90] The latter employed the above-mentioned bag experiment, while the other investigators operated upon duodenal fistulæ.

This experiment showed that the water drunk by the animals ran off almost completely out of the upper fistula, so that they perished in a short time, in spite of abundant feeding through the lower fistula. Therefore, a high duodenal fistula was established and the small intestine shut off in a manner analogous to that employed by the above-mentioned authors. The experiment of v. Mering demonstrated that the stomach *absorbs no noticeable quantity of water,* but that carbonic acid, alcohol, sugar, dextrine, peptone, and common salt are absorbed, the amount absorbed increasing with an increase in concentration of the solution. Accompanying the absorption of these substances is a more or less *active secretion of water into the stomach* (diluting secretion, H. Strauss), which increases in amount proportionately to the quantity of substance absorbed. This secretion of water, which was found by Jul. Miller[91] and H. Strauss[92] to occur also in man, seems to be of great significance, especially in case of poisoning (to which attention was called by Harnack); the water serving as a protective agent against the action of corrosive substances. Moritz and Brandl assume that the object of this dilution of solutions brought into the stomach was to facilitate their absorption in the intestines, since, for example, solutions of sugar are absorbed in the intestines only when greatly diluted. Brandl found also that solutions of peptone were absorbed from the stomach only in small quantities. He noted, for instance, that when 150 cubic centimetres of a 10-per-cent. solution of peptones were introduced into the stomach, not even 5 per cent. was absorbed at the end of two hours. This author also agreed with the above-mentioned investigators that alcohol was readily absorbed from the stomach; for instance, a 20-per-cent. solution of alcohol was completely absorbed at the end of two hours. Indeed he found that the presence of alcohol facilitated the absorption of other substances, as, for example, solutions of peptones. v. Mering adds in his article that the absorption of alcohol, carbonic acid, peptone, and common salt in the stomach resembles the physical process of diffusion. This view has existed for a long time as regards the so-called crystalloid substances (salts, sugar), and in more recent times has been considerably advanced by the progress of physical chemistry, especially the theory of osmotic pressure.

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[83] Tappeiner, Zeitschr. f. Biologie, 1884, Bd. 20, S. 52.

[84] v. Anrep, Reichert und du Bois' Archiv, 1881, S. 508.

[85] Smith, du Bois' Archiv, 1884, S. 481.

[86] Tappeiner, Zeitschr. f. Biologie, 1880, Bd. 16, S. 497.

[87] v. Mering, Verhandl. d. XII. Kongr. f. innere Medicin, 1893.

[88] Moritz, Verhandl. d. Naturforscherversammlung in Nürnberg, 1893.

[89] Hirsch, Centralbl. f. klin. Medicin, 1892, No. 47; 1893, No. 4, 18, 29.

[90] Brandl, Zeitschr. f. Biologie, 1893, Bd. 29, S. 277.

According to the researches of Winter,[93] Roth and Strauss,[94] Pfeiffer and Sommer,[95] the osmotic pressure of the gastric contents seems to be subject to a certain fixed form of regulation. At the height of digestion it is less than that of the blood. The osmotic pressure of the blood represents a lowering of the freezing point of $\Delta = -0.56$, that of the gastric contents (D), according to the researches of Strauss and Roth, a lowering of the freezing point of $\Delta = -0.38$ to -0.44, but it may at times be -0.32 to -0.55; the average being -0.40. It is abnormally high in the human stomach, as a rule, in the presence of lactic acid; excessively high freezing points are met with in occasional cases of superacidity, and low degrees exist in apepsia gastrica. The normal stomach has, then, the property of lowering the freezing point in solutions in which it is higher than -0.37, and, *vice versa*, of increasing it in solutions in which $\Delta = -0.32$. The greater the difference between Δ of the introduced fluid and D, the more rapidly and the more thoroughly does the degree of concentration become equalised. Of importance, furthermore, is the behaviour of the molecular concentration of the fluid which is introduced. In this connection Strauss distinguishes between gastroisotonic, gastrohypertonic, and gastrohypotonic solutions.* When a gastroisotonic fluid is introduced, no change occurs in its molecular concentration; but the introduction of a gastrohypertonic fluid causes a dilution, and a gastrohypotonic solution a condensation of the molecular concentration. A further discussion of these experiments, which need further exploitation, cannot be entered upon here.

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[91] Jul. Miller, Archiv f. Verdauungskrankheiten, 1896, Bd. 1, S. 233.

[92] Strauss, Zeitschr. f. klin. Medicin, 1896, Bd. 29, Heft 3 u. 4; Vergl. auch Roth und Strauss, Zeitschr. f. klin. Medicin, 1899, Bd. 37, S. 144.

[93] Winter, Archive de physiol., 1896.

[94] Roth and Strauss, Zeitschrift f. klin. Medicin, 1899, Bd. 37, S. 144.

[95] Pfeiffer and Sommer, Archiv f. experimentelle Pathologie und Therapie, Bd 43, S. 93.

The absorption of so-called colloid substances (albumen, gelatine, mucus, vegetable gum) must be regarded as *an active cellular process.* As regards the stomach and intestine, the lymphatic corpuscles of the adenoid tissue of the gastric and intestinal mucous membrane are concerned in the absorption, according to F. Hofmeister, whereas Heidenhain, with reference to the fats, clings to the theory that the free fat-drops are absorbed. Schilling[96] was also able, in examining the gastric mucosa of freshly-killed calves, to find fat-droplets partly within the cells, partly free between the cells, and partly within the epiploic and cœliac glands which drain the lymphatic vessels of the stomach. Substances absorbed from the stomach are carried to the portal system of the liver, where, after feeding with sugar, a much more decided increase of sugar was found than in the blood of the carotid arteries (v. Mering, Drosdorff). The albuminous bodies are at first peptonised, and then gradually changed again into albumens; the careful investigations of Fr. Hofmeister and Glässner[97] demonstrated that this conversion does not occur in the blood, but in the mucosa of the stomach and intestines themselves.

See tests for absorption for diagnostic purposes, in the Chapter on Chemical Examinations.

Motor Functions (Peristole, Peristalsis) of the Stomach.

The motor activity of the stomach has two functions, consisting primarily in the thorough mixture of the foods and their saturation with gastric juice, and secondarily, the elimination of the chyme, during which the two openings of the stomach alternately close and open. The first is accomplished partly by the motor activity of the stomach (see below), and partly by the passive movements transmitted to the stomach by other organs. Thus the diaphragm and abdominal muscles exert pressure during respiration, while the spleen and liver also exercise rhythmical compression upon the stomach. The impulse transmitted to the stomach by the heart is also not to be underrated. Finally, increased activity of the muscles of respiration also assists the motor activity of the stomach, although not to a very great extent.

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* Strauss considers a solution gastroisotonic, the Δ of which varies between —0.36 and —0.48°. Solutions Δ is greater than —0.48° are designated by him as gastrohypertonic, and solutions the Δ of which is less than —0.36° as gastrohypotonic.

[96] Schilling, Fortschritte der Medicin, 1901, No. 23.

[97] Glässner, Beiträge zur chemischen Physiologie und Pathologie, 1901.

Rotation of the stomach on its longitudinal axis has been described and considered for a long time as an important adjunct to the process of digestion. The investigations of Betz,[98] Lesshaft,[99] and Baum[100] failed, however, to demonstrate either active rotation of the stomach by contraction of the gastric musculature, or a passive rotation of the stomach on its axis. The only movement of the stomach, when that organ is filled, consists in a forward movement of the greater curvature (it being directed downward when the stomach is empty), accompanied by an elevation to a certain extent of the anterior gastric wall, and the lesser curvature is directed backwards.

Opinion is still divided as to the *active* movements of the stomach. It is assumed that when food is introduced, a tonic contracture of the stomach occurs for a short time. It gives place to the regular peristole one-half to an hour later. With reference to the latter, we know that waves travel at short intervals from the fundus toward the pylorus. Beaumont has already directed attention to the fact that this undulatory movement obtains quite considerable activity, which is manifested by circular bands at the pylorus (transversal band). Beaumont was able to show, by means of a thermometer bulb, which he introduced into the gastric fistula (of the Canadian St. Martin), that solid bodies are seized by the pyloric antrum and rolled into the pylorus, kept there for a short time, and then finally driven back again by means of anti-peristaltic waves into the cavity of the stomach, and then along the lesser curvature to the cardia, from where they pass into the fundus, to the greater curvature, and so on. Whether we assume the peristole to be as described above, or whether we accept the somewhat analogous observations made by Brinton,[101] so much seems certain, that active movements occur at the pylorus which are concerned in the thorough saturation of the chyme. Brinton compared the stomach with a cylinder into which a centrally pierced piston is pushed, which exerts pressure upon the peripheral strata in one direction, while the central strata pass in the opposite direction. The nature of the movement at the pylorus, its origin and by what stimulated, represent one of the most interesting and most disputed questions in the physiology of the stomach. The investigations of Hofmeister and Schütz[102] on stomachs excised from freshly-killed dogs go to show that two typical phases of gastric movements exist. The *first* begins with movement in the fundus, whose circular muscles contract from all sides. At the same time vertical contractures travel from the cardia to the pylorus, which are most marked on the greater curvature and at the border of the pylorus. The *second* phase of motor activity is confined exclusively to the pylorus. Beginning with a tight constriction at the border of the pyloric portion of the stomach, the longitudinal and circular musculature of the pylorus contracts so that this portion becomes much shorter and narrower; the movement finally ends with a vigourous contraction of the pyloric sphincter. After this phase is completed, it begins anew. Hofmeister and Schütz saw recurrent waves of contraction originating in the pyloric sphincter, as described by Beaumont, only when the pylorus contained solid particles; hence, according to the ancient theory of van Helmont, the pylorus possesses a sort of elective function. The above-mentioned observations were essentially confirmed by the more recent investigations of A. Hirsch[103] and Fr. Moritz,[104] and by the Röntgen-ray test of Roux and Balthazard[105] and Cannon.[106] The first author who experimented with high duodenal or small intestinal fistula in animals, reached the conclusion that the expulsion of chyme depends directly upon the digestibility of the ingested food. Thus, for example, while compact food remained in the stomach for some time, and was eliminated gradually in small quantities, the expulsion of liquids followed in a much shorter time. The ingestion of moderate quantities of fresh water hastened considerably the expulsion of the compact substances. These results agree exactly with those of Fr. Moritz. He observed, by means of experiments partly on himself and partly on animals with high duodenal fistula, that the fundus was capable of producing only weak waves of contraction, whereas the pylorus, on the contrary, presented stronger waves. In the fundus the foods are sorted in a way, so that the fluid constituents are advanced first. The movements of the stomach are otherwise rhythmic. Accordingly we distinguish in the stomach two divisions with reference to its motor activity, namely, the fundus of the stomach, which serves only as a sort of reservoir or digestion chamber (Fr. Moritz), and is not concerned or at least only very insignificantly so, in the expulsion of the chyme. The other division is the pylorus, to which the contraction waves are practically confined. Roux, Balthazard, and Cannon arrived practically at the same conclusions. These investigators studied the movements of the gastric contents in perfectly healthy animals, by administering food so prepared that it was opaque to the x-rays (bismuth subnitrate, cinnabar, and mercury), and then exposing the animals during digestion to the Röntgen rays. The shadow cast by the stomach appeared dark-gray or black on the screen, and changed constantly in a characteristic manner with respect to its form and gradually also in size. Worthy of mention in the researches of Roux and Balthazard is the study they gave to the influence of the peptones on the contractility of the pylorus. They found that aqueous solutions of peptones excited secretion and produced an increase in the number of gastric contractions. A similar effect is produced, only more slowly, by solutions of hydrochloric acid. Similar in many respects are also the observations of v. Openchowski.[107] According to him, a median groove is noted between the upper and middle third of the stomach, and occasionally somewhat lower, which persists until the stomach has ended its motor phase. The latter consists of a peristaltic wave, which runs regularly from the muscular groove to the pylorus, and which is most marked at the pyloric antrum. The upper third of the stomach, which borders on the cardia, does not participate in this phenomenon. The cardia and pylorus show special movements, close and open alternately, whereby the cardia closes when the pylorus opens. v. Openchowski attributes the control of this closing and opening mechanism to the presence of certain groups of ganglia cells within the serosa, which are connected with vagus and sympathetic fibres. Of interest as regards animal physiology, yet not without significance to human physiology, are the recent investigations of Grützner and Heyde.[108] These consisted principally in giving animals various kinds of foods in definite quantities and at regular intervals. The animals were killed and their stomachs removed *in toto* and placed in a freezing mixture. Sections of the frozen stomach showed, in an extraordinary manner, the peculiar layer-formation of the food, as well as the distribution of the acid or neutral reaction, provided the food had been previously mixed with an acid indicator. It was shown that, as a rule, the fresh food passes into the centre of the mass of old food, which, in turn, is pressed against the gastric walls. These innermost layers of food do not come in contact with the gastric juice, and hence retain their neutral or alkaline reaction for hours. The outer layers alone become slightly acidulated and digested, and are later separated from the remaining food, and propelled into the pro-pyloric portion of the stomach.

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[98] Betz, Prager Vierteljahrsschrift für praktische Heilkunde, 1853, S. 106.

[99] Lesshaft, Virchow's Archiv, 1882, Bd. 87, S. 69.

[100] Baum, Deutsche Zeitschrift für Thiermedicin und vergleichende Pathologie, 1889, Bd. 15, S. 401.

[101] Brinton, Lectures on the Diseases of the Stomach, London, 1864.

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[102] Hofmeister and Schütz, Archiv für experimentelle Pathologie, 1886, Bd. 20, S. 1.

[103] Hirsch, Centralblatt für klinische Medicin, 1892, S. 993.

[104] Fr. Moritz, Verhandlungen der 65; Versammlung der Gesellschaft deutscher Naturforscher und Aerzte in Nürnberg, 1893.

[105] Roux and Balthazard, Archive de physiologie, January, 1898.

[106] Cannon, American Journal of Physiology, May, 1898. [Observations on the Mechanics of Digestion, Journal of the American Medical Association, March 21, 1903.]

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[107] v. Openchowski, Deutsche medicinische Wochenschrift, 1889, No. 35.

[108] Grützner, 23. Versammlung der Balneologischen Gesellschaft, Stuttgart, 1902.

The diastatic ferment of the saliva may accordingly act for a long time, undisturbed by the acids. The left half of the stomach is so constructed that it may be looked upon as a sort of sugar-reservoir or storehouse (comparable to the auricle of the heart), which delivers the prepared material in small portions to the pro-pyloric portion or true stomach.

The results obtained by Rossbach,[109] in experimenting on dogs, present essentially different views. He found that when the stomach is filled, the peristaltic movements commence at first weakly, then gradually increase in strength, and persist from four to eight hours. The movements are confined exclusively to the pyloric half of the stomach; the fundus presents no motor activity whatever during the whole period of digestion, and is only slightly contracted around its contents. The movements of the stomach always begin at the same point, that is, about the middle of that organ, where they constrict it markedly; the contractures travel in the form of waves, within twenty seconds, to the pylorus, where they cease abruptly. The contraction waves constrict the stomach so markedly at the height of digestion that its lumen is annihilated. The peristaltic movements may be increased by the administration of not too large quantities of cold or warm drinks; large amounts of cold water stop, and narcotics inhibit the contractions, which is of therapeutic significance. The fasting stomach is small and withered in appearance, and contracts weakly and only rarely; as a rule, it presents no active movement. The pylorus is (according to Rossbach) tightly closed during the four to eight hours of digestion, and does not allow a single drop of gastric contents to enter the duodenum. The emptying of the stomach occurs suddenly and in repeated spurts. During digestion and fasting the sphincter of the pylorus is in such a state of tension that the stomach is shut off from the intestine. The duodenum presents no *active* movement during the whole period of digestion; nevertheless its secretion continues. Not until the pylorus opens do the peristaltic movements of the duodenum begin, and they continue just as long as chyme enters from the stomach. If the stomach ceases to work, the movements of the upper portion of the intestines also stop. Rossbach is furthermore of the opinion that a relationship exists between the stomach, the pylorus, and the duodenum; thus, the irritation of the foods upon the gastric mucous membrane causes, reflexly, at first a strong innervation of the pyloric sphincter and inhibition of the movements of the duodenal muscles; these phenomena are reversed later. The duodenal inactivity during gastric digestion permits the intestinal juices to collect, in order to neutralise the gastric juices.

The researches of Oser[110] call sharply to attention the contradictions existing amongst the different results of various investigators, and at the same time the difficulty of obtaining a definite solution of the problem of gastric peristalsis. Oser found that the nature of the pyloric closure varies in different animals, and even in the same animal during experimentation. Complete closure of the pylorus is favoured by the rapid flow of water into the stomach. Very low temperatures of the fluid seem to have a similar influence, whereas high temperatures fail to have such an effect.

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[109] Rossbach, Beiträge zur Lehre von den Bewegungen des Magens, des Pylorus und Duodenums, Deutsches Archiv f. klin. Medicin, 1889, Bd. 46, S. 296.

[110] Oser, Zeitschrift f. klin. Medicin, 1892, Bd. 20, S. 285.

Oser has furthermore studied very carefully the innervation of the stomach in animals which had been treated with curare. With reference to this subject, the author came to the following results: Spontaneous movements without *any regular rhythm* occur when the nerves are intact, as well as when severed. Animals are occasionally met with, whose stomachs remain at complete rest. Irritation of the vagus produces a marked contraction, which does not outlast the irritation to any noticeable extent, and which is followed by a dilatation of the organ lasting for a short time. Irritation of the vagus does not inhibit the course of the peristalsis when it exists before irritation is begun. Irritation of the splanchnics exercises a very weak contraction, but also a dilatation of the organ, which outlasts the irritation by a long time. The continuation of a spontaneous movement, existing before the splanchnic irritation, is inhibited. Frequently an after-effect of splanchnic irritation is manifested by increased peristalsis, which is much more active than before irritation. The increased peristaltic movements which set in as an after-effect of the irritation of the splanchnics can be stopped by splanchnic irritation. When the vagus and the splanchnics are irritated simultaneously, the motor-effect of irritation of the vagus is present at the beginning, but does not influence the continuation of the effect of splanchnic irritation. Irritation of the vagus is more or less ineffectual when the stomach has been brought at rest by irritation of the splanchnics; but the effect increases with the length of time intervening be-the continuation of the effect of splanchnic irritation. Irritation of the vagus is capable of inhibiting the peristalsis which arises as an after-effect. The vagus, as well as the splanchnics, are both active immediately during and after the irritation from a motor and inhibitory standpoint, only the relation between the motor and the inhibitory effect of the two nerves is different; that is, the motor function predominates in the vagus and the inhibitory function in the splanchnics. The vagus stimulation outlasts the irritation only a short time, while the activity of the splanchnics does not reach its highest point until after the irritation, and then in opposite forms, at first as inhibition and then as active peristalsis.

In spite of this apparently complicated arrangement, the process of gastric peristalsis proceeds mainly, as the researches of Hofmeister and Schütz, v. Openchowski, Hirsch, Moritz, and others have taught us, as follows: the emptying of the stomach occurs in a rhythmic manner and principally in the pyloric region. The liquid and pulpy substances are forced out first, while the solid bodies are still retained. Inasmuch as the pressure in the fundus of the stomach is very slight, only the easily-movable and watery constituents of the gastric contents are carried to the pylorus, on account of which a sort of selective arrangement is developed.

Of further interest is the factor which excites peristalsis. We may imagine that it is brought about by the mechanical influence of the ingesta, or by the increased congestion of the blood-vessels of the gastric mucous membrane. We have, however, never given up the view of Brücke,[111] namely, that the gastric acidity is the cause of the gastric movements. On the other hand, v. Pfungen[112] has promulgated the hypothesis that the alkalies cause the pyloric sphincter to open and that the acids cause it to contract. A. Hirsch[113] has undertaken to explain these contradictions. He found that in animals, in which duodenal fistulæ had been established, neutral as well as alkaline fluids pass in a short time from the stomach into the duodenum; likewise, also, dilute solutions of acetic acid. Solutions of hydrochloric acid (1 to 5 per mille) alone remained in the stomach a longer time. Stronger solutions of acids (it being immaterial whether acetic or hydrochloric acid is employed) caused vomiting. The closing of the pylorus in itself thus seems to be independent from the reaction of the gastric contents; not until the gastric mucous membrane has been over-irritated does active movement of the whole stomach begin (see Roux and Balthazard). This movement is then transmitted to the pylorus. The observations of Hirsch show that irritation of the stomach, when sufficiently strong, may cause the contractions to become so vigorous as to lead to a form of tetanus of the pylorus; the contractions, breaking against the tetanically-closed pylorus, become recurrent, the cardia and œsophagus become reflexly dilated, and the phase ends in vomiting.

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[111] Brücke, Lehrbuch der Physiologie, 4 Aufl., Bd. 1, S. 322.

[112] v. Pfungen, Ueber Atonie des Magens. Klin Zeit- und Streitfragen, Wien, 1887.

[113] Hirsch, Centralblatt f. klin. Medicin, 1893, No. 4.

DISEASES OF THE STOMACH

PART I

GENERAL DIVISION

I. METHODS OF GENERAL EXAMINATION

CHAPTER III.

The Anamnesis.

All clinicians and clinical text-books have so frequently emphasised the importance, in making a diagnosis, of an exhaustive anamnesis, which calls attention to the salient points and yet does not enter into details too minutely, that reference to its value might seem superfluous. However, the technique of the anamnesis, in diseases of the digestive organs, deserves an exhaustive discussion, for I have been repeatedly convinced that the elicitation of the facts and data which are concerned, has, as a rule, not received the attention and the accuracy required for a true conception of the disease.

The local anamnesis, which refers to the digestive apparatus and its functions, must be preceded, without exception, by the *general* anamnesis, which includes all previous diseases as well as hereditary conditions. Above all things, one should not allow oneself to be deceived, in making an unbiased examination, by the previously-mentioned (page 2) tendency of patients to lay special stress upon disturbances of their digestive apparatus. Hurried conclusions must be particularly avoided in this connection, because of the relationship so frequently existing between digestive disturbances and other organic diseases, and because of the purely symptomatic rôle played by the former in the symptom-complex of the constitutional diseases (tuberculosis, syphilis, diabetes mellitus, carcinoma, gout, leukæmia, chlorosis, pernicious anæmia, etc.).

The *local* anamnesis, which refers to special diseases of the alimentary canal, must take into consideration all points concerned in the diagnosis. The fewest sins of omission will be committed if the examiner proceeds as if the determination of the *status præsens* were impossible for any reason.

The following questions will serve better to elicit the points of importance to the anamnesis than any long-winded explanation:—

Age and sex?

When did the disease begin, and how long has it existed?

Did it begin suddenly or gradually?

In case of the former, under what circumstances did it begin, or what symptoms accompanied the onset (fever, chills, infectious diseases), or what were the causes (indigestion, sudden cooling off, trauma, fright, etc.)?

How did the disease manifest itself at the beginning?

Did the clinical picture undergo any changes since its onset, and what were they ?

Weight?

What is the nature of the complaint existing at present?

Of the above-mentioned questions the *age* and occasionally the *sex* play an important rôle. Thus carcinoma is a disease which appears, as a rule, beyond the third decade, whereas gastric ulcer occurs in the majority of cases at the time of puberty, and preferably in the female sex. On the other hand, carcinoma of the œsophagus develops far more frequently in men than in women. The *influence of traumatism* upon the stomach or its neighbourhood must not be disregarded. Thus, for example, cases of ulcer have been frequently described as due to an injury. Carcinoma can, of course, not be caused by traumatism; the latter may, however, hasten the growth of malignant neoplasms.

The *weight* is also of great practical importance. It is highly important, in regard to loss of weight, to distinguish between alimentary (artificial) or nosogenous loss of weight. The former is frequently a result of improper mode of living, or fear of eating which is attributable to true disturbances, or to a false conception of the easy or difficult digestion of articles of diet; or it may be the result of anorexia, which, again, may be psychical or organic in origin. The loss of weight in severe organic diseases of the stomach is, as a rule, also accompanied by a decided impairment of strength; whereas this impairment is often absent in cases of alimentary loss of weight or in functional disturbances. The loss of weight without other symptoms is not always an unfavourable sign, and likewise an increase in weight cannot always be looked upon as a favourable symptom. Even in carcinoma of the stomach (and intestines) the weight may show a marked increase temporarily (even without increase of ascites and œdema). The real strength is, however, usually not increased.

We had now better refer to the special disturbances, concerning which the following points are to be considered:—

A. How is hunger or appetite?

(*a*) Is it regular or not?

(*b*) Is the appetite completely lost?

(*c*) Or is it variable and freakish, and only present for certain substances?

(*d*) Is the appetite increased, or is there a spasmodic and impulsive, ravenous hunger?

(*e*) Is anorexia present, which disappears with eating; or *vice versa*, does a good appetite disappear after eating a few morsels of food?

(*f*) Does a temporary appetite exist which alternates with anorexia, or does a normal appetite, or even anorexia, change into bulimia?

(*g*) Does the feeling of satiation develop after eating, or does it not?

(*h*) Does repugnance exist toward food, against all or only certain articles of diet, especially meat and its products?

B. Is the thirst increased?

Does the amount of urine excreted correspond, or not correspond, to the quantity of water ingested?

C. How is the sense of taste?

(*a*) Normal, insipid, pasty, sweet, sour, unpleasant, or foul?

(*b*) Does the taste vary with the time of the day or hour?

D. What is the nature and time of the different meals?

(*a*) Approximate estimation of the quantity of the ingested foods or liquids?

(*b*) Especially with reference to proteids, carbohydrates, fats, salts, liquids (tea, coffee, cocoa, milk, water, mineral water)?

E. Are alcohol and tobacco abused?

F. Do disturbances of swallowing or symptoms of dysphagia exist?

(*a*) In the form of a globus in the throat?

(*b*) A feeling of resistance or foreign body (at what point)?

(*c*) Do pains arise in the region of the pharynx or œsophagus (of what character: regularly or irregularly, associated or not with eating, its intensity)?

(*d*) Does an obstruction interfere with the direct passage of food (at what point, and what is the state of the food which is obstructed (solid, semi-solid, pulpy, liquid)?

(*e*) Has bleeding from the pharynx or œsophagus been observed by the patient or determined by a physician?

G. Is a feeling of pressure present in the region of the stomach?

(*a*) At what point, and where felt most intensely?

(*b*) Only after eating, or independent of the ingestion of food?

(*c*) Does the pressure begin immediately after eating (exact answers desired), or after the lapse of some time?

(*d*) How long does this pressure persist (for hours or only during digestion and then disappear again)?

(*e*) Do the quality and quantity of the food exert any influence upon the onset and the intensity of the pressure?

(*f*) Does this sensation of pressure disappear for days or weeks at a time, only to recur again later? Can the patient give any causes for these variations (dietetic errors, excitement, etc.)?

H. Is a sensation of fulness present after eating?

(*a*) In the epigastrium, or lower down, or all over the abdomen?
(*b*) Also after small meals, or only after the principal meals?
(*c*) Or does it not occur until during the digestion and in direct association with ingestion of food?
(*d*) Is the feeling of fulness influenced by the quality or quantity of the foods and liquids (meat, vegetables, fats, fluids)?
(*e*) Does this sensation appear also at other than meal times?

I. Are pains felt in the region of the stomach?

(*a*) In connection with the meals, or independently of them?
(*b*) In case of the latter, do they appear at regular intervals of hours, days, weeks, months, and always in the same intensity and same character (see under *e*)?
(*c*) At what point are the pains localised (describe approximately), that is, within a circumscribed area, region, or in a division of the abdomen, or do they change their location?
(*d*) Are they confined to the area involved, or do they radiate in various directions? If so, describe the latter.
(*e*) Character of the pains (boring, griping, gnawing, sensation of a sore, burning, heart-burn, cramp-like); can the patient attend to his work in spite of them, or must he remain in bed? How long do the pains continue (seconds, minutes, hours)? Do they begin anew after intervals of rest?
(*f*) *Are the pains permanently or temporarily relieved by the ingestion of food (which food); are they exacerbated, or do they remain uninfluenced?*
(*g*) If they are provoked by the introduction of food, which has the most influence in this respect, a liquid, a soft, or a solid diet?
(*h*) Are the pains less when at rest, and do they increase or not increase in severity on motion?
(*i*) Does pressure upon the painful area lessen or increase the pain?

K. Eructation (belching).

(*a*) Slight or troublesome?
(*b*) Quietly or loudly audible (explosive)?
(*c*) In connection with digestion, or also when the stomach is empty?
(*d*) Character of eructation: odourless or odourous (like rotten eggs, fæculent; like acids, greasy, rancid)?

(*e*) Is it accompanied by the simultaneous regurgitation of food (taste, odour, and condition of the latter)?

(*f*) Duration of the period of eructations (for minutes, or shorter or longer)?

(*g*) Do they occur only occasionally, or regularly every day?

L. Vomiting and nausea.

(*a*) Is the nausea protracted or temporary, and is it associated with eating?

(*b*) Is it relieved by rest?

(*c*) Character of the vomitus: food, mucus, bile, acids; quantity, colour, and odour?

(*d*) Does the vomiting occur daily, and, if so, at what time?

(*e*) Does the vomiting set in when the stomach is empty and fasting, or after eating, and, if so, how long after eating?

(*f*) *Was blood or any other abnormal constituent ever present in the vomitus?* If blood was present, what was its quantity and colour (light or dark red); was it mixed with food, was it foamy? (Compare also with *M.*)

(*g*) Is the vomiting periodical (with periods of normal health), and what is the length of the intervals?

(*h*) Describe the act of vomiting (previous sense of pressure and fulness, nausea, feeling of choking, and severe cramp-like pains)?

(*i*) Is the act of vomiting preceded by a feeling of discomfort, or are the food-substances ejected without such abnormal sensations?

(*k*) Is the vomiting followed by relief?

(*l*) Is the vomiting frequently repeated during the day or night, and at what intervals of time? Does it occur whenever food is taken?

M. Evacuation of the bowels.

(*a*) Is constipation or diarrhœa present?

(*b*) In case of the former, are the bowels at any time evacuated spontaneously, and at what intervals of time?

(*c*) If not, what remedies are employed (purgatives [state which], injections, enemata, suppositories), their character and results?

(*d*) Appearance, colour, odour, admixture with abnormal elements (mucus, membranes, blood, pus, entozoa, tar-coloured stools)?

(*e*) Are the stools accompanied by pain; if so, where is it located?

(*f*) If diarrhœa exists, how frequent are the evacuations and what is the composition of the stools (excrements, or blood, or pus, or a mixture of them)? What is the consistency of the fæces?

(*g*) Is the diarrhœa accompanied by abdominal pain and rumbling?

(*h*) Is the diarrhœa continuous or periodical?

- (*i*) Is the diarrhœa frequently or regularly preceded by constipation, and is the former always followed by a renewed period of constipation?
- (*k*) Is tenesmus present constantly or occasionally? Does the tenesmus lead to evacuation? What is the composition of such evacuations (see under *f*)?
- (*l*) Do hæmorrhoids exist? Is there an excretion of blood, mucus, or similar material from the anus?
- (*m*) Is there a feeling of soreness or burning? Has the anus been the seat of fissures, fistulæ, proctitis, or periproctitis?

N. Flatulency.

- (*a*) Its character and associated symptoms (marked abdominal distension, unpleasant sensations, headache, anxiety, palpation, dyspnœa)?
- (*b*) Does it only follow a certain form of diet, or all varieties of food?
- (*c*) Length of duration and time of occurrence (as regards length of time after eating, short or long period of duration, regular or periodical; if the latter, are the intervals typical or irregular)?
- (*d*) Borborygmi, rumbling, gurgling, and griping?

O. Subjectively perceptible peristaltic movements.

- (*a*) In what part of the abdomen?
- (*b*) Dependent upon or independent of the digestive period?
- (*c*) Regular or periodical?

P. Sensation as if a foreign body were present in the abdomen.

- (*a*) Location; does its position change with the body motions?
- (*b*) Does pressure cause pain? Do pains arise spontaneously in the region of this sensation?
- (*c*) Has the supposed foreign body increased in size?

The diagnostic value of some of these points of anamnesis is undoubtedly clear and therefore requires no further discussion, whereas some of the other facts stated above must be considered more fully. We shall discuss them in the order followed above.

We must distinguish between *appetite* and a *sensation of hunger*. The latter is felt in the stomach, and its presence may be manifested by considerable pain, even though the appetite is absent. Appetite is the precursor of hunger, and consists of an inclination or longing for food (M. Rubner). The opposite to appetite is repugnance, and its worst form, disgust for food. Both conditions occur physiologically, but then only temporarily (for example, surfeit of food, poorly-cooked foods, anger, etc.). If it is a persistent condition, it is either the expression of nervous affections, or of severe organic gastric disease, or of other pathologic processes.

Obstinate loss of appetite is, for example, frequently observed at the beginning of pulmonary phthisis. A thorough examination of the lungs should therefore not be neglected in every case of obstinate anorexia.

The reversed condition of the appetite, that is, a morbid increase of appetite, is designated as *bulimia* (the hunger of oxen), or as *cynorexia* (the hunger of dogs). Bulimia is, in the majority of cases, a concomitant symptom of nervous disturbances; in other cases it may appear also as a complication of nervous or organic gastric and other diseases (gastric ulcer, tape-worm, sexual diseases, affections of the brain, neurasthenia, diabetes mellitus, etc.).

Bulimia must also be distinguished from *polyphagia* and *acoria*. In the former an abnormal quantity of foods and liquids is ingested without any noticeable increase in appetite; this may be the result of abnormal metabolism *or an insufficient feeling of satiety*. This state is called *acoria*. Polyphagia is most frequently observed in diabetes, in the convalescence from severe diseases (typhoid, intermittent), and also in diseases of the brain (epilepsy, hydrocephalus, cerebral tumours, etc.).

Various forms of changes in the appetite exist between the above-mentioned extremes (*parorexia*). It is markedly affected by external and psychical influences, but in the majority of cases it is a symptom of gastric neuroses.

The appetite is generally lessened in *cancer, atrophy of the gastric mucous membrane,* in *chronic gastritis in its severe forms,* and in *amyloid degeneration*. The appetite is occasionally entirely absent in these conditions, or it exists only for substances which are, as a rule, prohibited (generally acid and highly-seasoned foods). A repugnance and even disgust for meat and other albuminous substances exists in nearly all cases of cancer. Normal appetite, or even a voracious appetite, is observed only rarely in cancer.

The appetite varies considerably in *gastric dilatation,* depending upon the cause and the severity of the disease, as well as upon the condition of the chemical processes. A normal appetite, and occasionally a voracious appetite, occurs in hyperchlorhydria; a poor, and occasionally entire absence of, appetite occurs, as a rule, in ectasia accompanied by marked stagnation.

The appetite remains generally unaffected in *gastric ulcer,* and indeed a morbidly increased desire for food may exist, especially during the painless intervals. In rare cases, however, the feeling of hunger may also be lessened. This must, however, be distinguished from the fear of food [sitophobia] because of the pain it causes. The state of the appetite in the sequelæ of gastric ulcer (pyloric stenosis, dilatation, carcinomatous ulcer) undergoes gradual change, and is similar to that manifested by these conditions under other circumstances.

The appetite is quite variable in *gastric neuroses*. The principal type of this form of appetite consists in the disappearance of the desire for food after beginning a meal, which had been preceded by an excellent appetite; a feeling of satiety steps in "after the first spoonful of soup," and the stomach feels full and like an extremely heavy weight. In other cases the appetite acts in a capricious and queer manner, depending upon the mood, it being exaggerated sometimes, and reduced to nil at other times. Periods of good appetite alternate with intervals of lessened and even absent appetite. A number of variations of the most peculiar nature occur in this connection, the enumeration of which would, however, require too much space. Common to all is the protean type, which jumps suddenly to extremes and which is almost positively indicative of the nervous origin of the disease.

A different form of appetite, which vacillates within wide limits, is manifested by such disturbances of digestion as accompany or follow other chronic diseases, such as pulmonary phthisis, malaria, heart-disease, and nephritis. A definite type of this form of appetite cannot even be approximately given, for the nature and extent of the causal diseases have a marked influence upon the digestive functions.

Finally, I wish to call attention to the fact that conclusions as to the condition of the gastric functions cannot be deduced from the state of the appetite. I have repeatedly found that the gastric functions were perfectly normal, although the appetite was completely lost, and, *vice versa*, I have, on the other hand, seen a large number of cases in which the appetite was unaffected in spite of marked functional disturbances.

The condition of *thirst* is of great importance in diseases of the stomach. It is only exceptionally diminished, and remains normal in many gastric affections. Thirst is markedly increased in diseases of the stomach associated with loss of gastric motility, especially in gastric dilatation. These cases present also the diagnostically very important antagonism between the quantity of water ingested and the amount of urine excreted. Thirst and diuresis are not rarely increased in gastric ulcer and in hyperchlorhydria. The thirst is likewise increased in many cases of gastric neuroses.

The condition of the *taste* and the odour of the breath are of limited value in the diagnosis. These conditions may both be normal in severe gastric affections, and, on the other hand, markedly altered in mild gastric disturbances. The taste is frequently insipid, pasty, and bitter in chronic gastritis and in carcinoma, foul in marked fermentative processes and ulcerating gastric cancer, as well as in decomposition within the intestines. The taste may also be changed qualitatively in nervous dyspepsia. On the other hand, it is usually normal in ulcer and in all other gastric disturbances associated with superacidity. A bad taste and foul breath are, however, caused, as a rule, by diseases of the mouth, pharynx, and tonsils. Carious teeth, periodontitis, inflammations of the alveoli and of the pulp, and tonsillar plugs (thrombus) (J. Herzfeld,[1] B. Fränkel,[2]) also occasion a stale, foul, or bitter taste and odour. The last-mentioned author calls attention, and justly so, to the fact that the foul odour occasionally exists in the mind of the patient, and not in reality. According to my experience, uncleanliness of the tongue is an important cause of the bad taste and odour. By scraping off the fur of the tongue (particularly the dorsum of the tongue), with a cotton swab or a spatula, one may frequently convince oneself (and the patient) as to the source of the bad odour.

The *composition and the number of meals* may give information which is of diagnostic value. Reference to the *diet* is especially useful for the determination of the state of nourishment, as well as the possible loss or increase of body weight (see above, page 54). The quantity and variety of liquids are also of importance. Large quantities of liquids, whether in the form of soup, coffee, tea, mineral water, or alcoholic drinks, easily tend to cause relaxation of the gastric musculature, even though, as we learned in our student days, the stomach shows a wonderful adaptability in this respect. *It is of practical importance that the old routine method of ordering soups, as it is still practiced by some physicians, leads frequently to an exacerbation of many gastric affections, and particularly to a loss in weight.*

The patient's statements as to *disturbances of swallowing* are of little use unless corroborated by objective examination; they may, however, serve to a certain extent as a guide to the diagnosis. At first the location of the dysphagia must be examined. The extent of the obstruction is furthermore of significance, and may be readily judged from the sort of food ingested by the patient at the time (solid, semi-solid, liquids). We must not neglect to ask the question whether disturbances of swallowing are present only temporarily, that is, does the œsophagus show normal permeability for days or hours at a time. It must also be determined whether the spasm or similar disturbance is independent of the ingestion of food. *Œsophagism* is to be taken into consideration under these circumstances.

The regurgitation of food or liquids is of diagnostic significance when a *diverticulum* is suspected. Yet too much reliance must not be attributed to these signs. Patients are occasionally able to call the attention of the physician to a certain swelling in the throat—the typical site of a high œsophageal diverticulum—which appears after taking food.

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[1] J. Herzfeld, Therapeutische Monatshefte, 1897, Januarheft.

[2] B. Fränkel, Archiv für Laryngologie, 1900, Bd. 10, Heft 1; ref. nach Therapie der Gegenwart, 1900, Bd. 2. S. 80.

The *"globus hystericus"* can hardly be overlooked in carefully eliciting the anamnesis, yet it is probably not superfluous to call attention to a sensation similar to the "globus hystericus" which is caused by tenacious mucus adhering to the pharyngeal or œsophageal wall. *Pains* occasioned by the passage of the bolus, in the absence of stenosis is indicative of simple and inflammatory processes in the œsophagus or in its neighbouring structures (mediastinum, lymphatic glands, etc.). The origin of hæmorrhage in cases of dysphagia cannot be proven, as a rule, by the anamnesis, and therefore requires further examination.

Pressure in the epigastrium is a symptom shared in common by many diseases of the stomach. It is therefore only rarely of diagnostic importance in differentiating between the various affections (see below). In the mild cases a slight and tolerable pressure develops shortly after eating, which disappears in the course of digestion, whereas in the severe forms of gastric disease the pressure continues for hours; in still other cases the pressure does not develop until the height of gastric digestion is reached. Finally, patients are met with who constantly suffer from a feeling of pressure or fulness in the epigastric region, even when the stomach is empty or after the ingestion of minute quantities of food. This sensation of pressure is frequently caused by *swelling of the liver,* but may also be due, in individual cases, to nervous affections of the stomach, to constipation, hæmorrhoids, etc. This pressure is frequently, but not always, accompanied by a *feeling of fulness.* The latter is also associated with the former in the majority of cases, whereas, on the other hand, pressure may occur independently of the feeling of fulness. It is of as little diagnostic value as is epigastric pressure; in drawing conclusions, however, a marked feeling of fulness is usually referable to an abnormally long sojourn of the ingesta in the stomach, and hence to a disturbance of the motor mechanism in the stomach as well as in the intestines.

Pains in the region of the stomach are of unusually great diagnostic significance, provided time is taken to determine exactly their location, intensity, character, and time of occurrence. It is of primary importance to determine whether the "stomach-ache" really originates in the stomach. Even intelligent patients constantly refer pains in any part of the abdomen to the stomach. It is well, therefore, not to place too much reliance upon these assertions, and we must think besides of true cardialgia, of cholelithiasis, nephrolithiasis, intestinal colic, herniæ in the linea alba, and pancreatic colic, and we have to examine for them.

If the patient's history shows that gastric pain is probably present, it becomes necessary to determine whether it bears any relationship to the ingestion of food. Such a connection is found to a certain extent in both organic and nervous affections of the stomach, but most frequently in the former. It is, furthermore, of interest that the course of a large number of chronic affections of the stomach may be unaccompanied by pain. This is the case, for instance, in the various stages of the majority of the chronic gastric catarrhs (in its stricter sense) and in many nervous affections of the stomach. Gastric cancer may also, unless situated directly at the pylorus, run a painless course, or at least manifest no severe pain. Carcinoma of the pylorus, on the other hand, is accompanied, as a rule, by pain during its advanced stages; its absence is exceptional.

The pain varies in *myasthenia* and in *gastric dilatation;* mild forms of these conditions may exist without any pain whatever. Pains of more or less severity do not develop unless marked retention of the gastric contents exists, especially when accompanied by advanced fermentative processes.

Pain is rarely absent in *gastric* or *duodenal ulcer.* However, gastric ulcer may run a course which is practically free from pain; this has been proven by accidentally finding an old ulcer-scar at autopsy, and also by the sudden perforation of a peptic ulcer without any previous manifestations of that condition. The pain of a gastric ulcer has a definite character, which serves, as a rule, to distinguish it sharply from the other pains occurring in the same location. *It is gnawing and burning in nature, and feels as if due to a sore spot.* If cramps occur in the stomach, the pain will radiate either diametrically, or laterally backward toward the dorsal vertebræ. In rare cases the vertebral pain is more pronounced than the epigastric. In *gastric ulcer* the pain develops from thirty to forty-five minutes after the ingestion of food, whereas in duodenal ulcer the painful seizure does not begin until much later (about ninety to one hundred and twenty minutes) after taking food. In the latter case the starting point of the pain is in the left parasternal line, whereas in ulcer of the stomach it is most frequent in the median line. The patient does not always suffer from intense pain in gastric ulcer, but instead complains of a dull or painful pressure. Rest on the back lessens the pain of gastric ulcer, while motion of the body and occasionally even changing of the posture and mechanical influences (lifting, carrying, riding) increase it.

Similar to, if not identical with, the above pain are the painful attacks of a severe form of *hyperchlorhydria.* The latter may, as a rule, be distinguished from the pain of ulcer by the fact that the administration of albumen (milk, meat, eggs) regularly abolishes it for a short time. On the other hand, hyperchlorhydria need not necessarily in itself cause severe pain (physiologic hyperchlorhydria).

Of great diagnostic and prognostic interest are the periodic gastralgias which occur in the form of *gastric crises* in tabes dorsalis, or as a forerunner of the same, or more rarely as independent neurosis (v. Leyden,[3] Rémond,[4] Boas,[5] Vagedes,[6] and others). The attacks consist of cramp-like contractions of great severity in the epigastrium, which radiate downward, as well as to the left hypochondrium as far as the spine. These attacks are accompanied by copious vomiting, which is prolonged for days and even weeks. *Attacks of this nature which occur at variable intervals may be looked upon, in the large majority of cases, as initial symptoms of tabes, provided no other causal disease exists which explains this symptom-complex.*

Associated with this subject is *gastroxynsis* (Rossbach[7]). This likewise consists of severe pains occurring suddenly in the midst of perfect health and at irregular intervals, which are accompanied by a feeling of soreness and *corrosion* of the gastric wall and by intense headache and vomiting.

Still to be mentioned is the so-called heart-burn, or *pyrosis*. This may be due to the action of strongly-acid chyme regurgitating into the œsophagus (pyrosis hydrochlorica, Sticker[8]), or it may be readily caused by the gases (ammonia, etc.) evolved during the digestive process, and finally it may be attributed to organic acids. Pyrosis may, however, be present, as has been shown by McNaught, when the stomach contains a normal amount of acid. Ewald,[9] with whom v. Leube[10] agrees, considers heart-burn as a form of motor neurosis, due to an irritative state of the motor sphere.

The nature of the disease may ocasionally be surmised from the character of the *eructation*. If continuous, spasmodic, and tasteless, it may be looked upon without doubt as a manifestation of a nervous disease of the stomach (eructatio nervosa). The eructation may, moreover, be sour; the latter being due to hydrochloric as well as organic acids, especially lactic and butyric acid and other volatile fatty acids. Only when strongly acid masses regurgitate into the mouth, which may set the teeth on edge, is it permissible to suspect hyperchlorhydria. On the other hand, foul eructations which smell like *hydrogen sulphide* or other fœtid eructations point toward a high grade of fermentative or putrefactive processes in the stomach. The origin of the eructation is also, under certain circumstances, not without significance. As I have frequently proven, the source of the eructation is occasionally the intestines, and not the stomach. Worthy of mention is the fact that many patients, because eructation is popularly considered a sign of good health, practice it as a "sport," on account of which the cardia may gradually become relaxed.

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[3] v. Leyden, Zeitschrift f. klin. Medicin, 1882, Bd. 4, Heft 4, S. 605.

[4] Rémond, Archiv général. de médecine, 1889.

[5] Boas, Deutsche medicinische Wochenschrift, 1889, No. 42.

[6] Vagedes, Inaug-Diss., Berlin, 1891.

[7] Rossbach, Deutsches Archiv für klin. Med., 1884, Bd. 35, S. 383.

[8] Sticker, Münchener med. Wochenschr., 1886, No. 32 u. 33.

[9] Ewald, Klinik der Verdauungskrankheiten II., 3 Aufl., S. 505.

[10] v. Leube, Spezielle Diagnose der inneren Krankheiten I., S. 240.

The patient's statements as regard *nausea,* and especially *vomiting,* are of great significance, and indeed, under certain circumstances, the diagnosis may be made from them alone.

Nausea occurs in a large number of gastric diseases, and it represents a common symptom in the various forms of chronic gastric catarrh. It occurs also in gastric cancer, accompanied by vomiting, or independently of the latter. It is, moreover, a frequent manifestation of the presence of *tapeworm.* Nausea develops likewise in many of the gastric neuroses; it is also a common symptom, either alone or accompanied by vomiting, in myasthenia and in gastrectasy with processes of fermentation. Nausea is also a common occurrence in anæmia and its associated dyspeptic disturbances. As a rule, nausea, or malaise, is present immediately upon eating, although it may not set in until the height of digestion is reached. Nausea, when the stomach is empty, develops frequently in gastric neuroses, especially in bulimia, hysteria, at the menopause, etc., and in superacidity of neurotic origin; in the latter case, the nausea rapidly disappears upon the ingestion of food.

Vomiting is of even greater importance than nausea.

It must, of course, be determined primarily whether true vomiting or some other similar condition is concerned. The latter refers to the following: (1) *Regurgitation,* in which food is forced into the mouth without nausea or spasmodic contractures of the stomach, and which, as a rule, is spit out. (2) *Rumination* (merycism), in which liquids or foods are forced upwards without causing any disagreeable sensation; these are then swallowed a second time (sometimes without re-chewing them) if they possess a pleasant taste; but, on the other hand, they are spit out if sour or unpleasantly bitter in taste (peptones). This process, which is probably attributable to a palsy of the cardia, has nothing in common with vomiting, and must be sharply distinguished from it. (3) Still other forms of vomit-like conditions are met with which are likewise unaccompanied by nausea, and are therefore similar to the "throwing-up" of children. Such is the vomiting in severe bronchitis, and is seen mainly in phthisical and emphysematous subjects. The majority of the cases of so-called reflex vomiting belong to this class (sexual diseases, biliary and renal calculi, pharyngeal catarrh, etc.). Finally, the various forms of so-called nervous or hysterical vomiting need to be mentioned. These forms are distinguished from typical vomiting by the absence of the preparatory stage of the vomiting act (yawning, pallor, general depression, and a choking sensation); for that reason the attack does not cause the patient much disturbance, and indeed it may even bring relief. In *true vomiting* the symptoms of which are commonly well known, we must take into consideration its *onset* and the *appearance*, the *quantity*, the *odour*, and *the taste* of the vomitus. With reference to the onset of the vomiting attack, the time of occurrence and its relationship to the ingestion of food are of great diagnostic importance. A drinker vomits in the morning the masses of mucus which have accumulated during the night; the sufferer from superacidity and gastrochylorrhœa* vomits in the morning or at night the excess of gastric juice which is produced even in an empty stomach. The vomiting of pregnancy after breakfast is well known.

In contradistinction to the above is the vomiting following the taking of food. *The frequent occurrence of vomiting after eating is a serious symptom, provided gastric neuroses can be excluded, and is indicative of organic changes in the gastro-enteric tract.* It occurs, as a constant condition, in gastric or duodenal ulcer, in processes accompanied by ulceration of the follicles, in myasthenia, in gastric dilatation, in chronic gastric catarrh, in gastrochylorrhœa, in gastric cancer, as well as in tumours in the neighbourhood of the stomach and small intestines, and in stenoses of the latter whatever the cause may be.

The vomiting in *ulcus ventriculi* occurs, as a rule, shortly after the meal, and generally in connection with the typical ulcer-pains.

Vomiting occurs in *myasthenia* as well as in *gastric dilatation,* generally at the height of digestion, when the disproportion between the amount of ingesta and the expelling force becomes manifest. Hence, in gastric dilatation vomiting during the course of the night is not an uncommon occurrence. The vomiting is also preceded in this case by more or less severe pain.

In *chronic gastric* catarrh, vomiting likewise does not develop until one or two hours after meals, and is preceded by a sensation of pressure and nausea; the vomiting may, however, set in occasionally at the conclusion of the meals. Characteristic of this disease is, moreover, the repeated vomiting at intervals, which is followed by relief for some time.

In *superacidity,* vomiting does not set in, as a rule, until after the lapse of several hours, when it follows the increased formation of acid and the development of spasmodic contractions of the pylorus (pylorospasm); vomiting at night is also a common phenomenon in this condition.

Vomiting in *cancer* depends upon the location of the tumour (pylorus, lesser curvature, greater curvature, fundus) and upon the dynamics of the expelling force. Vomiting is one of the commonest symptoms in carcinoma of the pylorus, whereas, on the other hand, it may be completely absent or only moderate in degree in cancer of the walls of the stomach. Inasmuch as the need for food in cancer is slight, as a rule, vomiting is generally less frequent and regular than in a benign stenosis of the pylorus due to gastric ulcer. Thus the vomiting may not occur until a day after the last ingestion of food, and occasionally it may not appear until even later. However, with the increase in the stenotic phenomena and the spreading of the cancer to the gastric musculature, the vomiting may at times occur repeatedly each day and soon after each meal.

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*[I am using the term "gastrochylorrhœa" instead of gastrosuccorrhœa, in order to eliminate the Latin word succus from the otherwise Greek compound.]

Vomiting likewise does not occur in *stenosis of the small intestines* until the later stages of digestion are reached.

The quantity of the vomitus, although the patient or his attendants can estimate it only approximately, may offer important information as to the nature of the disease. Large amounts of vomitus are always indicative of marked peristaltic obstruction (consequent to pyloric or duodenal stenosis, adhesions of the pylorus with other sections of the intestinal tract, or insufficiency of the musculature). A small quantity of vomitus is nearly always a symptom of chronic gastritis; the amount may also be small in gastric ulcer, although from personal experience fairly large quantities may be observed in that condition.

The *appearance* of the vomitus is occasionally of diagnostic value, although little reliance can be placed on the judgment of the laity in this respect. Of most importance is the presence of abnormal constituents—blood (fresh or altered), bile, mucus, saliva.

Blood which flows over the surface of the stomach is altered very rapidly and becomes dark with the formation of more or less compact clots. Dark red masses of uncoagulated blood are vomited only when the hæmorrhage is very abundant; in that case we must exclude hæmorrhage from the œsophagus (œsophageal ulcer), passive congestion (cirrhosis of the liver, valvular heart disease, etc.), or specific disease of the blood-vessels of the œsophagus or stomach (varicosities). If the blood is retained in the stomach for a longer period of time, its colouring-matter becomes converted into the well-known coffee-grounds or chocolate-coloured mass. The admixture of minute quantities of blood, when acids are absent, does not cause a change in the colour of the vomitus or gastric contents (occult hæmorrhage). For its detection and significance, refer to the chapter on Examination of the Gastric Contents.

Bile and *pancreatic juice* are frequently mixed in the vomitus, and are indicative of severe vomiting or the termination of repeated vomiting spells, especially when the stomach is empty. *Continued vomiting* of bile is a manifestation of an obstruction in the *passage of the descending portion of the duodenum*. In stenosis of the upper portion of the duodenum, on the other hand, the appearance of the vomitus is similar to that of pyloric stenosis.

The presence of large quantities of *mucus* points toward gastric catarrh, although the admixture of that constituent may also be attributable to the preparatory stage of the act of vomiting, as well as to a chronic pharyngitis, bronchitis, etc. The same holds true for *saliva.*

The *odour* and *taste* of the vomitus cannot, as a rule, be looked upon as of much importance. When, however, the odour is fæcal, search should be made for intestinal obstruction. The taste may be stale, bitter, putrid, or sour. The vomitus is stale in taste in chronic gastritis because of the presence, in large amounts, of mucus or saliva; bitter, when a marked quantity of peptones or bile is present; decomposed or foul, when fermentation exists in the stomach; and sour, in hyperchlorhydria or gastrochylorrhœa, or also in the presence of organic acids.

Disturbances in the *movement of the bowels* are frequently met with in diseases of the stomach, partly dependent and partly independent of them. Thus constipation exists nearly always in advanced forms of gastrectasia. Habitual constipation is also the rule in marked hyperchlorhydria and supersecretion. On the other hand, diarrhœa is frequently present in chronic gastritis or achylia. In other forms of gastric disease the bowel movements are variable. Intestinal disturbances, namely, habitual constipation, may, however, lead secondarily to gastric derangement. Especial attention was called to this fact very recently by Knud Faber.[11] It frequently becomes necessary also, in diseases of the stomach, to gain information as to the exact state of the bowels, partly through the anamnesis and partly by direct investigation.[12] [Schmidt.[13]]

Flatulency accompanies diseases of the stomach as well as of the intestines. The time of its occurrence frequently yields useful information as to the cause of the flatulency. Thus, flatulency developing soon after eating points toward the abnormal evolution of gas in the stomach, whereas its occurrence after digestion is completed is referable to fermentation in the intestines. An observant patient is occasionally able to state whether the quality of the ingesta (vegetables, pastry, fats, etc.) has any influence in this respect. The periodic occurrence of flatulency, or such as occurs at irregular intervals independently of the character of the food, favours a nervous origin.

Rumbling and gurgling indicate abnormal fermentative processes in the intestines, the cause of which cannot, however, be determined without careful examination.

Peristaltic movements in the abdomen, which are *subjectively perceptible,* may occasionally be recognised and described as such by the patients.

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[11] Knud Faber, Archiv für Verdauungskrankheiten, 1902, Bd. 8, Heft 1 u. 2.

[12] Compare Boas, Diagnostik und Therapie der Darmkrankheiten, 2. Aufl., Leipzig, 1901. [Translation, Dr. Basch, Appleton & Co., New York.]

[13] Adolph Schmidt, The Test-diet (translation by Charles D. Aaron), F. A. Davis Co., Philadelphia, 1906.

The latter experience a sensation as if a worm were slowly moving to and fro (whence the very appropriate term "vermiform movement"). The exact location of these processes, which are abnormal in every case, must be determined by further examination. For greater detail refer to *Inspection.*

Many patients, who are suffering from an abnormal tumour, experience a certain *sensation as if a foreign body were present.* It certainly occurs more frequently in movable tumours, which rise and fall with respiration, yet it is not so rarely noted by patients with tumours of the stomach. The patients may furthermore describe approximately the site, the size, the painfulness, the rate of growth, etc. Too much diagnostic importance must, of course, not be attributed to these statements, although they may be used with advantage, yet cautiously, in the determination of the *status præsens.*

CHAPTER IV.

The Examination of the Patient.

Inspection.

Although *inspection* does not play so important a rôle in the diagnosis of internal disease as do other methods of examination, yet under certain circumstances it is a factor not to be underestimated; it represents one of the most difficult expedients, and can only be mastered after long training and experience. Inspection must take into consideration every factor which is in any way concerned in the diagnosis of an internal disease. For the accurate diagnosis of a disease of the digestive system, some particularly important factors are to be considered, a knowledge of which is indispensable. The first of these is a *careful inspection of the oral cavity.*

Inspection must furnish the following information: 1. Condition of the teeth and the gums. 2. Appearance and condition of the tongue. 3. Abnormalities of the palate. 4. Condition of the pharynx and also of the naso-pharyngeal space.

The appearance and condition of the teeth may suggest information, in as far as carious teeth, gingivitis, or stomatitis may readily cause or predispose to disease of the stomach. Likewise, the failure of proper mastication when teeth are deficient may be productive of an obstinate and prolonged irritative condition of the stomach. Any artificial teeth which may be present should also be carefully examined, for if not properly fitted, they may produce a chronic irritation of the gums and of the mucosa of the mouth, which in turn is followed by unfavourable effects upon gastric digestion. Furthermore, as every experienced practitioner knows, unclean teeth create conditions favourable for the breeding of micro-organisms which may interfere with the digestive process, partly by causing local irritation, and partly by their entrance into the stomach. Miller,[1] in his excellent and oft-quoted book, gives an instructive description of this subject, which is so much neglected in practice.

The *appearance and the condition of the tongue* are, in my opinion, still too highly valued in comparison with the above subject. I am able to state, according to my experience with many cases of gastric and intestinal diseases, that although the diagnostic significance of the appearance of the tongue is not wholly worthless, yet it is only of secondary importance. I have seen patients with "coated" tongues, who possessed an enviable appetite, and, as was demonstrated by means of the tube, a perfect gastric digestion; whereas, on the other hand, I have known patients with absolutely clean tongues who suffered from anorexia for weeks and months. Particularly noticeable is the smooth condition of the tongue in gastric cancer.* The condition of the tongue is in general (excepting acute febrile diseases) nothing else than an *expression*, or, better, a *counterpart of the state of the oral or pharyngeal cavity*. Careful examination of the latter two will usually show that the cause of the bad tongue is either caries of the teeth, periodontitis, gingivitis, stomatitis, pharyngitis, tonsillitis (tonsillar plugs), swellings in naso-pharyngeal space, adenoid vegetations, salivation, or an altered condition of the saliva, etc. The tongue is also frequently coated, in case of an absolute milk diet independently of any alteration in the stomach. Not until all of these conditions have been excluded does it become possible to connect the change in the tongue with disease of the stomach. But even in that case I feel justified, according to investigations on this subject, in coming to the conclusion, primarily, that in chronic digestive disturbances the appetite and condition of the tongue apparently bear no direct relationship to each other, and secondarily, that absolutely no decision can be formed as to the nature and degree of the gastric disturbance from the condition of the tongue.

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[1] Miller, *l.c.*

Our knowledge of the pathogenesis of a coated tongue has been greatly advanced by the recent researches of Johannes Müller[2] and Hugo Fuchs[3]. Müller and Fuchs were the first to make the astonishing observation that no less than 62 per cent. of all healthy individuals in youth and middle age presented coated tongues. On the other hand, this was only observed in 33 per cent. of persons of old age, this being evidently due to the development of lingual atrophy in advanced years. Disease of the oral cavity (caries of the teeth, stomatitis, pharyngitis, etc.) existed in 66 per cent. of young persons whose tongues were coated; in 34 per cent. the mouth was intact. In all of the cases in which the tongue was markedly coated, the filiform papillæ were considerably elongated and arranged closely together. These, together with a horny layer which is considerably thickened, wrinkled, and overgrown with bacteria, have a decided influence upon the coating of the tongue; this coat is further increased when the tongue is kept insufficiently clean. The tongue is coated in sick individuals whenever it is not cleaned mechanically, that is, in persons who are able to talk but little, who ingest only liquids, and who cannot swallow well. In my opinion, the desiccating influence of the fever which destroys the epithelium must also be considered. The microscopical picture of the coating of the tongue varies only in the healthy and the sick, in so far that in the latter the scraped-off fur contains a far greater abundance of liquids and morphotic elements than the former. The leucocytes were greatly increased in sufferers from lung and heart disease and from cancer of the stomach. According to Fuchs, the former method of relying upon the pathognomonic significance of a coated tongue should by all means be discontinued.

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* Johannes Müller gives as follows the percentage relationship existing between coated tongue and diseases of the stomach: Acute gastritis, 82 per cent.; chronic gastritis, 55 per cent.; gastric ulcer, 69 per cent.; carcinoma of the stomach, 65 per cent.; nervous dyspepsia, 68 per cent.

[2] Johannes Müller, Münchener medicin. Wochenschrift, 1900, No. 33.

[3] H. Fuchs, Ueber den Zungenbelag und seine Bedeutung, Inaug-Diss., Würzburg, 1898.

In the *soft palate*, the tonsils (size, scars, inflammatory processes, ulcers) are principally to be considered, inasmuch as they may increase the secretion of saliva and thus lead to disturbances of digestion. The *pharynx* is of special importance, chiefly because it is frequently chronically inflamed in smokers and alcoholics. Since chronic disease of the stomach is not rarely a sequel to protracted pharyngitis, this relationship cannot be too carefully observed, both diagnostically and therapeutically. I have seen cases of "gastric catarrh," treated for some years, recover in a surprisingly short time after curing the pharyngeal catarrh.

Inspection of the *abdomen* itself, to be sure, offers but meagre results in many cases; in others, however, quite useful information may be obtained.

Before proceeding with inspection of the abdomen, we must make clear what knowledge can be obtained in that manner. *Under favourable circumstances*, namely, when the stomach is abnormally distended, or in advanced stages of emaciation, we may note the outline of the stomach, especially its lower anterior surface; and when the stomach has descended, the lesser and greater curvatures may be seen, as well as the contour of a single intestinal coil or even of whole sections of intestines. Furthermore, abnormal and more or less sharply outlined projections of certain portions of the abdominal wall may be recognised, or, especially when the abdominal walls are thin, neoplasms may be observed and their size and limits estimated to a certain extent. When sunlight or lamplight, collected by means of a converging lens, is reflected from the direction of the head of the patient upon the anterior abdominal wall, a number of delicate shadows will be perceived which will be of considerable assistance when palpation is practised.

A very characteristic picture is produced by herniæ of the abdominal wall, especially in case of herniæ of the *linea alba* (epigastric hernia). These vary in size (lentil-seed to hen's egg), are either multiple or single, and may, under certain circumstances, cause considerable pain and vomiting, although occasionally their course is unaccompanied by symptoms. The hernia consists partly of omentum and partly of small incarcerated fatty growths (also called preperitoneal lipoma*).

The opportunity is frequently offered to observe *peristaltic movements* in the stomach and the intestines. They may be noted independently of any exciting cause, or following external irritation (ether-spray, faradisation, percussion-hammer, etc.). In the region of the stomach they run in the direction of the physiological peristalis, although in rare cases antiperistaltic movements may also be observed (as, for example, in intestinal stenoses or in high-grade pyloric stenosis). When the abnormal movements of the stomach are marked and occur independently of the ingestion of food, we speak, according to the precedent set by Kussmaul,[4] of "peristaltic unrest" of the stomach. It occurs in difficult expulsion of the ingesta from the stomach, and in that case is always indicative of pyloric stenosis; or it is a form of motor neurosis (tormina ventriculi nervosa). I had the opportunity to observe for a long time two cases of *nervous peristaltic unrest of the small and large intestines,* with simultaneous constipation and pains in the region

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* For further information on this subject, refer to Part II, page 634.

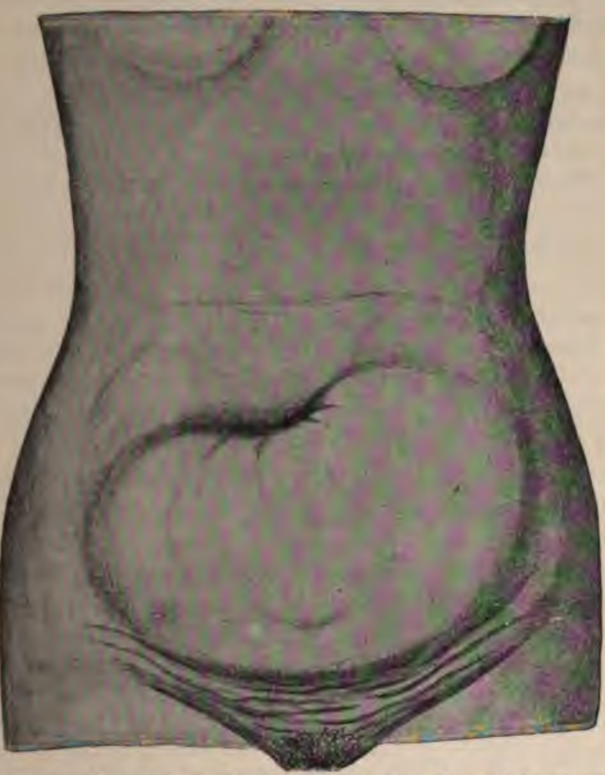

Fig. 10.—Visible Stiffening of the Stomach in a Case of Carcinomatous Stenosis of the Pylorus (personal observation).

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[4] Kussmaul, Die peristaltische Unruhe des Magens, etc., Volkmann's Sammlung klinischer Vorträge, 1880, No. 181.

of the intestinal canal.[5] Aside from the truly visible gastric peristalsis, a form of marked gastric contraction exists, as has already been observed by Cruveilhier,[6] which, analogous to the nomenclature adopted by Nothnagel for the intestines, I designated as *"gastric stiffening"* (Magensteifung[7]). By palpating a *full* stomach with the hollow of the hand, one may occasionally feel that organ rise and gradually become hard and stiff, which the patient may recognise every now and then in the form of more or less severe pain; in a few seconds, frequently accompanied by distinctly audible gurgling or clucking sound (glouglou, of the French), or with more or less active eructations, the stomach may be felt to collapse again. These tonic contractions may recur rapidly in pronounced cases, or may be observed only after a considerable length of time has elapsed. Occasionally only an increased tonus (Fig. 10) is observed, or, better, felt in place of or in association with the stiffening of the fundus of the stomach. As has already been emphasised by Cruveilhier, gastric stiffening is of extraordinarily important diagnostic significance in the presence of pyloric stenosis; the latter may be either pylorospasm or a true organic stenosis. The degree of stiffening represents an excellent criterion of the degree of stenosis. Tonic contractions have been observed not only in the fundus, but also in the pylorus (Schnitzler,[8] Hartmann,[9] Rütimeyer,[10] Brunner,[11] and others); in nervous individuals they assume the form of a spasmodic tumour, although they are also seen in organic stenosis of the pylorus. I observed such a case (stenosing pyloric cancer) several years ago. Similar observations have also been made by Bouveret,[12] who called attention to the intermittent distension of the right epigastrium as an early symptom of pyloric stenosis. Pædiatric literature in particular is filled with observations on such spasmodic pyloric tumours.

Eichhorst[13] states that he had recognised in several cases during life the existence of an hour-glass constriction of the stomach, in a low position at about the middle of the greater curvature, which was corroborated at autopsy. I also had a case with evident hour-glass constriction of the stomach, but without post-mortem.

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[5] Boas, Verhandlungen des 15. Kongresses für innere Medicin, 1897, S. 479 u. f.

[6] Cruveilhier, Traité d'Anatomie pathol. générale, 1852, Bd. 2, S. 857.

[7] Boas, Deutsche medicinische Wochenschrift, 1902, No. 10. Consult also Rütimeyer, Archiv für Verdauungskrankheiten, 1901, Bd. 7, H. 1 und 2.

[8] Schnitzler, Wiener medicinische Wochenschrift, 1898, No. 15.

[9] Hartman, cited from Rütimeyer.

[10] Rütimeyer, *l. c.*

[11] C. Brunner, Beiträge zur klinischen Chirurgie, 1901, Bd. 29, S. 520.

[12] Bouveret, Lyon med., 1901, No. 13.

[13] Eichhorst, Lehrbuch der physikalischen Untersuchungsmethoden, Bd. 2, S. 167.

For inspection of the stomach after inflation with carbonic acid, see page 105.

Palpation of the Stomach.

Palpation of the stomach is as difficult a method of examination, as it offers valuable diagnostic information. It is quasi the quintessence of all diagnostic explorations, and therefore indispensable. To be sure, it is not performed with the mere "Hineingreifen ins volle Menschenleben," but, instead, it is absolutely necessary to *think while palpating*, and to *palpate while thinking*.

As may be easily understood, the technique of palpation cannot be learned from description, but depends more or less upon personal training. Furthermore, the technique of palpation varies considerably amongst the different examiners. Thus, for example, many physicians palpate with flat fingers, and others with curved fingers; many consider that drawing up of the knee makes palpation of the abdomen easier, whereas others do not. Nevertheless, in the course of years, some general approved principles have been adopted. I will incorporate these with my own experiences in the following manner:—

1. A thorough palpation requires, as a rule, that the alimentary canal be empty; in many cases, however, a moderate fulness of the stomach and intestines does not interfere with palpation. When the fulness is marked, or when the abdomen is distended from gas or large amounts of contents, it is absolutely necessary to empty the stomach (by the passage of a tube) and the intestines (by irrigation).

In a few cases, as in examination of gastric (and intestinal) stiffening, (see page 74), the technique of palpation is reversed; that is, a full stomach is preferred to an empty one.

2. In palpation the hands should be comfortably warmed, for touching the skin with cold fingers is not only unpleasant to the patient, but it increases the difficulty of the procedure, on account of the contraction of the abdominal wall, which arises reflexly.

When, however, it is desired to determine the presence of gastric stiffening, better results are obtained only by means of a cold hand, or by spraying with ether.

3. Palpation must be practised when the patient is in the dorsal position, for only in that manner sufficient relaxation of the abdomen can be obtained; occasionally, to be sure, it is wise to compare the results of palpation in the dorsal with those obtained in the erect posture, or palpation may be completed in the latter position. As a rule, however, palpation of the parts which are at all accessible to that method can be performed with the greatest degree of certainty and distinctness in the dorsal posture. Occasionally the lateral is to be preferred to the dorsal position (as in tumours of the fundus), to insure better results.

4. In order to secure the necessary degree of relaxation it is well to draw the patient's attention from the object to be examined, by asking questions on the history of the case, or such as bear no relation to the real disease.

5. Relaxation is also increased, in some cases, by flexing the knees or the hips, although in other cases this procedure is of little use.

6. Short and rapid breathing has nearly always a favourable influence.

7. A method to be recommended in all abdominal tumours consists of bimanual palpation, and is performed in such a manner that one (best, left) hand presses upon the tumour being examined, so that the other hand is free to palpate it conveniently. I have found that in some cases it is useful, by exerting strong pressure upon the back, to raise deep-seated neoplasmata toward the palpating hand.

8. When sufficient relaxation is obtained, it is at first highly important to make a brief study of the topography of the abdominal organs. Accordingly, beginning in the epigastrium, palpate in the following order: The mesogastrium, the hypochondrium, the umbilical region, the iliac fossa or the ileo-cæcal region, the hypogastrium, and the inguinal region. In doing so, take heed as to the presence of pain; get information as to the condition of the hernial openings; bear in mind any abnormal resistance; also pay attention to the presence of any acoustic phenomena (splashing, gurgling), or new growths and other abnormalities.

9. Next, again perform a systematic palpation of the most important abdominal organs. It is best to begin by palpating the gastric region, at the same time paying attention to the transverse colon; next, palpate the liver and the region of the gall-bladder; then proceed downward along the colon, palpate the small intestines, and, passing to the descending colon, palpate the region of the spleen. Because of the frequency of dislocated or floating kidneys, it is wise, when palpating the corresponding portions of the colon, to take these organs into consideration in every case.

10. Palpation of the abdomen in women must be followed by examination of the genitals whenever organic disease is suspected.

11. In both men and women, in whom the least suspicion of rectal disease exists, an examination of the rectum should be made, to ascertain the presence of fissures, proctitis or periproctitis, anal fistulæ, hæmorrhoids, ulcers, or new growths. *When blood, mucus, or pus is excreted from the rectum, rectal exploration must not be neglected under any circumstances.*

According to observations of Chlapowski, Schuster, Lennhoff, and myself, palpation in a warm bath is occasionally followed by better results than by the ordinary method.

The method of Lennhoff[14] seems to me to be the most satisfactory, and consists in placing the patient on a sheet in a manner similar to the permanent bath. This sheet is held by means of bands, which are attached to metal clamps; the latter extend over the edge of the bath-tub like hooks, and are movable from side to side. By means of a simple mechanism the sheet can be raised or lowered at will. As a rule, it is most convenient when fastened about 20 to 25 centimetres below the upper edge of the bath-tub.

If, by the above-described methods, an anomalous condition be found present, further careful examination is required. For this purpose the following conditions are to be considered:—

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[14] R. Lennhoff, Berliner klinische Wochenschrift, 1896, S. 1073.

(a) *Sensitiveness to Pressure, Pain, Points of Pressure.*

The exact site and extent of the sensitive area are next determined, and in doing so information is obtained as to the part of the abdominal cavity involved. Sensitiveness in the epigastric region is not always referable to the stomach, for inflammation and swelling of the left lobe of the liver, and perhaps also of the pancreas, may reveal pain upon pressure in this area. Tenderness in the region of the greater curvature likewise does not, by any means, point toward involvement of the stomach in every case, for it is as likely to arise from the transverse colon, the small intestines, or the omentum. Unless the stomach has descended, the exact localisation of the source of the pain is impossible, when referable to the pylorus and the pyloric region, as well as the neighbourhood of the lesser curvature, because these structures are both covered by the liver.

From a diagnostic point of view, an essential difference exists between tenderness and pain, direct, circumscribed or diffuse, and algetic points partly in the region of the stomach and partly on the back. We must first distinguish between hyperalgesia of the skin and that of the structures lying immediately beneath. According to Head,[15] the internal organs are, to a fairly large extent, non-sensitive to various stimuli, and the localisation of pain in them is practically impossible; the stimuli are, however, transmitted by way of the sympathetics to a distinct spinal segment, whence they are referred to the sensory fibres of the skin which pass into the same segment. Inasmuch as the ability to localise is far better developed in these areas, the pain is referred, quasi through a psychical error in judgment, to the body surface instead of to the organ which is really diseased. The theories promulgated by Head require further confirmation; thus, Moll van Charante,[16] for example, in his dissertation, arrived at conclusions which deviated decidedly from the above. On the other hand, Hänel[17] investigated Head's researches and practically confirmed them, although he does not consider the method of differentiating between ulcer and functional diseases of the stomach to be practicable.

Sticker[18] observed still another form of hyperæsthesia in hysterical subjects, in which the sensitive zone is confined within the area of the diseased organs.

The presence of *tenderness on pressure* is demonstrated by the fact that the patient, on palpation of the epigastrium (the part generally concerned), experiences an unpleasant sensation, which, although it cannot be exactly defined, is not pain.*

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[15] Head, Die Sensibilitätsstörung der Haut bei Visceralerkrankungen, Deutsch von W. Seiffer, Berlin, 1896, Hirschwald.

[16] Moll van Charante, De hyperalgetische zonen van Head, Diss. Leiden, 1900; Ref. nach Centralblatt für innere Medicin, 1900, S. 251.

[17] Hänel, Münchener medicinische Wochenschrift, 1901, No. 1.

[18] Sticker, Zeitschrift für klinische Medicin, 1896, Bd. 30, S. 61.

This tenderness is a frequent but not constant symptom of various forms of chronic inflammation of the gastric mucosa, including chronic gastritis in its different stages, gastric neuroses, hypertrophy of the pyloric musculature, cancer which spreads out and is not far enough advanced to be palpated, etc. Tenderness on pressure may also occur in gastric ulcer (see below), especially in the later stages and when the scar-formation is not completed, or in the presence of adhesions, etc.

There is something so characteristic about *the true circumscribed chronic* pain that it can be clearly distinguished from all other forms of gastric pain. This pain is "sore," burning, and extremely unpleasant, so that when palpated the patient tends to resist. A very frequent sign of the typical gastric pain, which I have had the opportunity to regularly demonstrate to my audience, is *the painful twitching of the face, even when the involved region is touched with care.*

Occasionally, but by no means always, when the painful spot is touched, attention is called to a painful point lying diametrically opposite on the spinal column; in a few cases, indeed, the painfulness in that region exceeds that in the epigastrium. This type of tenderness, when pronounced and sharply localised to the gastric region, represents a valuable diagnostic sign in favour of gastric ulcer or hæmorrhagic erosions. It must not be assumed, however, from this that less severe pain excludes the existence of ulcer.

Acute or chronic inflammatory exudates or adhesions between the stomach and sections of the intestines may indeed cause tenderness similar to that of ulcer, but in such cases the ætiologic factor (traumatism, peritonitis, perigastritis, phlegmon of the stomach, previous operations, etc.) must always be demonstrable. Under favourable circumstances the differential diagnosis may be determined between ulcer of the stomach and that of the duodenum by the site of the ulcer pain. Thus, for example, it is highly probable that the ulcer involves the duodenum when the tenderness is constantly to the right of the median line, that is, in the parasternal line, and when spontaneous pain is located at this point, and furthermore when, in place of the blood-vomiting, blood is discharged in the characteristic form only from the rectum. This differentiation, furthermore, which, as has been mentioned, can only be determined with a fair degree of accuracy under favourable circumstances, is practically of no great significance, for the clinical phenomena in the acute stage, as well as the sequelæ, and finally also the therapy, vary from those of gastric ulcer only in very unimportant respects.

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* The beginner should note that every abrupt pressure exerted in the region of the stomach is not only unpleasant to the sick, but may simulate pathological conditions. It should not be forgotten that the epigastrium, especially in thin persons, is sensitive upon strong pressure, even in normal subjects.

Diffuse pain, that is, such as radiates over the whole epigastric region of the stomach, even as far as the umbilicus, occurs in the following conditions: 1. In phlegmonous gastritis. 2. In perigastritis. 3. In a perforating adhesion of the stomach with neighbouring organs, namely, transverse colon or pancreas and the liver, the duodenum, etc., and, finally, also in rupture of an ulcer into the peritoneal cavity. The first is distinguished from ulcer by fever, marked symptoms of collapse, and abdominal distension; the latter, aside from other signs, is distinguished by the development of the severest forms of painful paroxysms, resembling the picture of acute peritonitis, which are brought on by the slightest touch, and also by marked symptoms of general disturbance (see Part II, page 447). In favour of *perigastritis* is tenderness on pressure over the whole epigastrium, or the occurrence of a severe pain when the left lower thoracic wall is pulled forward, and it is particularly indicative of the above condition when it appears on vigourous stretching or bending backwards. The diagnosis at the best, however, can only be suspected from the symptoms.

Painful *points on pressure* are not uncommonly observed on the anterior abdominal wall; in my opinion, they are distinguished by the fact that they are far less severe, situated more deeply, and are not represented by corresponding points in the spinal column. The sensitive points which are most frequently taken into consideration lie in the region of the cœliac or solar plexus (surrounding the cœliac artery), and of the plexus of the abdominal aorta. According to J. Ch. Roux,[19] they are produced by gastric affections, as well as central depressor impulses.

Multiple circumscribed points of tenderness are frequently observed, of which a few lie even outside of the limits of the stomach. In such cases we may in all likelihood exclude ulcer, and in the other cases the other factors in the diagnosis must be considered. Since, in the presence of multiple algetic points, we have principally to deal with the question of nervous dyspepsia, and inasmuch as this condition is also simultaneously accompanied by other functional anomalies of the nervous system, it is rarely difficult to come to a conclusion, provided these symptoms are carefully considered. It must not be forgotten, however, that gastric ulcer may also occur in neurasthenics or in nervous dyspepsia.

The pressure-points on the spine, to which I first called attention, are of no little diagnostic importance (see Fig. 11). Of these we may distinguish: 1. Pressure-points, or, better, areas, in gastric ulcer. 2. In cholelithiasis. 3. In gastric neuroses. In gastric ulcer, in which they are pres-

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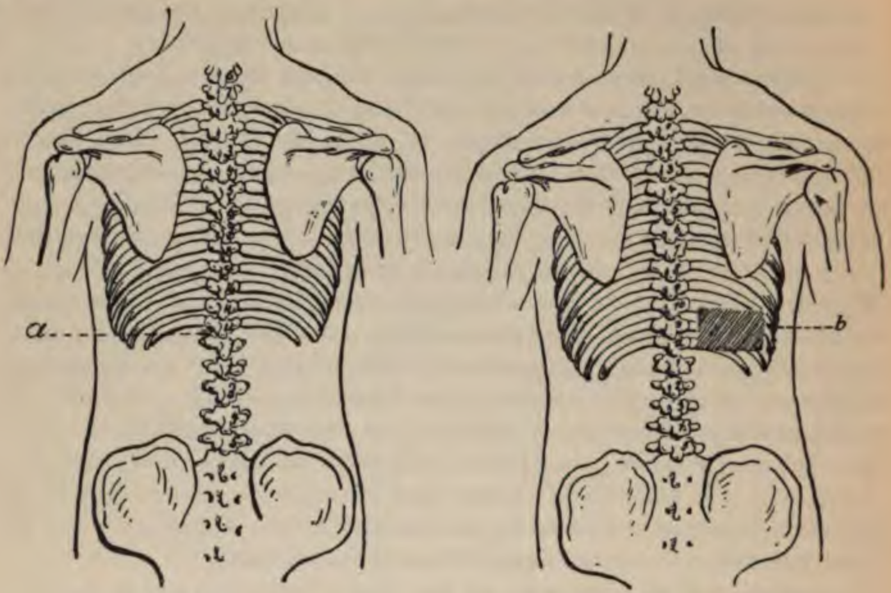
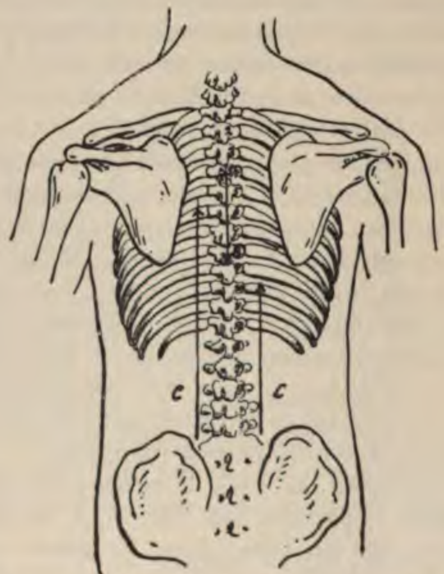
[19] J. Ch. Roux, Revue de médecine, 1899, S. 878.



a, Typical Pressure-point in Gastric Ulcer.

b, Typical Pressure-zone in Cholelithiasis.



c, Pressure-areas in Nervous Gastric Affections.

Fig. 11.

ent at least in one-third of the cases,* *they are located to the left of the spine, close to the body of the twelfth dorsal vertebra.*

This position may vary, however, which is not to be wondered at, considering the changes in the location of the stomach and of the ulcer; thus the painful area may be higher, at about the tenth or eleventh dorsal vertebra, or lower, as far down as the first lumbar vertebra. Sometimes a corresponding tenderness is situated to the right side, yet in that case the left one is the more sensitive; occasionally the point is located only in the right side, close to the vertebral body. This is particularly true when the ulcer is located at the pylorus or in the duodenum. As far as I can see, the dorsal point of tenderness bears no relationship to the zones of Head, as was claimed by Hänel. It is easy to demonstrate, for instance, that the dorsal pressure-points are not of the nature of hyperalgesia of the skin, for the pains are not elicited until deep pressure is exercised. The dorsal pressure-point seems, therefore, to be directly referable to the posterior gastric wall. According to Kelling,[20] on the other hand, the pressure-point described is nothing else than the supersensitive posterior branch of the intercostal nerves, and is due to reflex hyperæsthesia from the sympathetic nerves.

The point of tenderness on pressure in *cholelithiasis*,[21] which we discuss here because of the frequent consideration of the differential diagnosis between diseases of the stomach and the gall-bladder, is likewise located in the region of the twelfth dorsal vertebra, but at a point two or three finger-breadths distant from the vertebral bodies. It spreads from this point to the right, and in some cases as far as the posterior axillary line; it extends, therefore, over the whole posterior surface of the liver. No algetic points whatever are found in the left side. This tenderness may persist for weeks, or even months, after the attack of the disease, and therefore may be of assistance in doubtful cases, in which the swelling of the liver has already disappeared.

In contradistinction to these distinctly outlined local pressure-points, a large variety of painful zones are met with in *gastric neuroses;* thus they are found in some cases on the right, in other cases they are more pronounced on the left; or they occur in the form of single disconnected zones, or they are more diffuse and include a large portion of the back; sometimes they border on the bodies of the vertebræ, then again they are located more laterally. The three diagrams (Fig. 11, *a*, *b*, *c*) illustrate the types of the three different forms of pressure-points or zones along the vertebral column.

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* According to Pariser (Deutsche medicinische Wochenschrift, 1902, No. 15), they are decidedly more common, a view which coincides with my experience of the last years.

[20] Kelling, 74. Versammlung Deutscher Naturforscher und Aerzte in Karlsbad, Wiener medicinische Wochenschrift, 1902, No. 48.

[21] Boas, Münchener medicinische Wochenschrift, 1902, No. 15.

Since a few authors either absolutely ignore these practical and extraordinarily important pressure-points, or consider them to be only of slight importance, I must, after having proved their value for years, continue to give them the prominent attention they deserve. It does not follow that, because they may be absent or indistinctly developed, they are of no clinical use; for in these respects they resemble practically all clinical signs. Careful examination will rarely fail to reveal their presence in typical cases of ulcer and cholelithiasis; in other cases they occur at least frequently enough to support a doubtful diagnosis, or even to make a diagnosis possible.

In order to measure the intensity of pain of a certain section of the gastro-intestinal tract, I have devised an instrument called the "algesimeter."

The apparatus (see Fig. 12) consists of a hollow cylinder, which contains a spiral spring. A scale, showing whole, half, and quarter divisions, is attached to this cylinder, which indicates the degree of compression of the spiral spring, that is, from 0.5 to 10 kilos. An indicator placed around the cylinder moves with the piston, so that when compression is exerted the amount of pressure upon the handle can be immediately read on the scale. In order to exactly cover the painful area, the apparatus is furnished with three different-sized removable plates, which can be easily attached to its lower end.

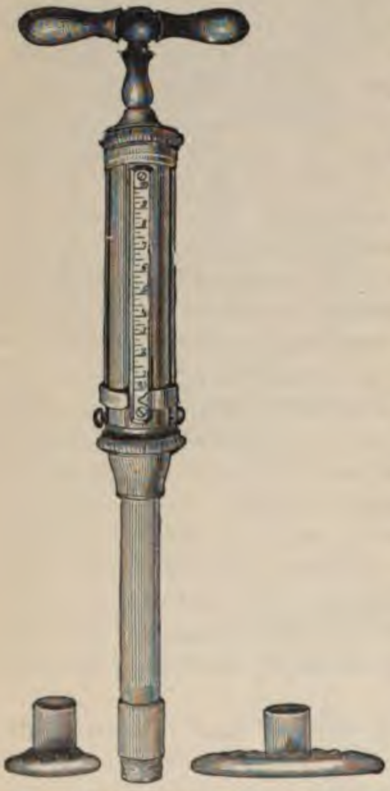

Fig. 12.—Boas' Algesimeter.

The following results have been obtained in estimating the degree of tenderness in the epigastrium. A normal stomach presents a degree of tenderness on pressure varying from five up to ten kilos. The lowest values are obtained in gastric ulcer, *in which they vary between 0.5 to 3 kilos.** If the tolerance for pain is higher than these figures, we may, as a rule, exclude gastric ulcer. Next to be considered is cancer, or, more correctly, carcinomatous disease of a portion of the stomach; in such cases the tolerance, when measured with the algesimeter, equals, on an average, from two to four kilos, and sometimes a little more. The highest results are obtained in chronic gastritis and in nervous dyspepsia, in both of which there is either no deviation from normal or only a slight fall below four to five kilos. It is also of importance to test the dorsal pressure-points. Normally, the sensitiveness to pressure is always over ten kilos, whereas in gastric ulcer the tenderness on the left, in the neighborhood of the twelfth dorsal vertebra, measures as low as three kilos. *Testing the pressure-points is not only of diagnostic, but also of therapeutic importance, for thus an objective control is obtained of the advancement in improvement and recovery of the patient.*

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* Pronounced tenderness may also occur in acute gastritis and gastro-enteritis, yet this disease is so distinctly characterised by its course and other symptoms that a false conception is inconceivable.

(b) Neoplasmata, Abnormal Resistance, Adhesions.

In studying new growths of the stomach it should be remembered that many different forms may originate in the gastric walls (fibroma, myoma, lipoma, papilloma, sarcoma, adenoma, tubercle, carcinoma), but that the latter of these is so frequently present that from a clinical point of view it is practically the only one to be considered. The researches of Brinton, Lebert, Luton, Ebstein, Katzenellenbogen, and others on the cadaver, all of which give practically the same results, show that the pylorus is involved in about 60 per cent., the lesser curvature in 17 per cent., the cardia in 8 per cent., the anterior and posterior wall of the stomach in 5 per cent., and the greater curvature in 2 per cent.*

It is furthermore remarkable that in about one-quarter of all cases both the stomach and the liver are infiltrated. According to Brinton's statements, the growth is most frequently scirrhous in nature (about 75 per cent.), that is, fibrous carcinoma (Faserkrebs), whereas the gelatinous variety (colloid and alveolar carcinoma) occurs only in 2 to 8 per cent. of cases. The medullary, the villous, and the telangiectatic forms of cancer are even less common.

Scirrhous cancer accordingly predominates in the region of the pylorus, so that when reference is briefly made to cancer of the stomach, the fibrous form at the pylorus is meant.

As in the case of every tumour, so also in new growths of the stomach, we take into consideration the *location, size, consistency, painfulness, boun-*

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* Examination of living subjects proves that carcinoma originates more frequently in the lesser curvature than in the pylorus, a fact which I already emphasised several years ago, and which has been recently demonstrated as indisputable by R. Borrmann in his studies on the abundant material in v. Mikulicz's clinics.

daries, and relationships to neighbouring organs, as well as the mobility and relationship to respiration.

If, for the above-mentioned reasons, the rarer forms of gastric new growths are disregarded, the *position* of the cancer changes with that of the stomach itself. As a rule, it lies in the triangle formed by the arch of the ribs and the umbilical line, yet it may not rarely be also found lower down; thus, for example, it may occur several finger-breadths below the umbilicus, or it may extend beyond the median line to the right or left, in which case it may occasionally be discovered on a level with the umbilicus, or even below it.

The *size* of the growth is likewise very variable. It may be entirely circumscribed and the size of a walnut or apple; it may be regular in form, or irregular; or it may consist of a cord-like or a flat cancerous mass. In advanced cases, with relaxed and emaciated abdominal walls, the contour of the stomach may be felt to consist of hard, dense, and nodular prominences (cancer *en cuirasse*).

The *consistence* of malignant growth is, as a rule, hard and dense. Its surface, as has already been stated, is frequently irregular, uneven, and nodular; the hardness of the tumour is more readily felt in some than in other parts.

Painfulness of the tumour is rarely absent, yet it varies in different individuals from moderate tenderness on pressure to marked sensitiveness which is not much less intense than the pain of gastric ulcer.

It should be noted that in certain cases it is quite possible to find a *movable* tumour. In that case the stomach itself is markedly displaced, so that its natural supports no longer answer their purpose; it is generally displaced downward.

The tumour may occasionally be intergrown with other organs, such as the liver, pancreas, duodenum, transverse colon, etc., which, having a strong influence upon the mobility of the tumour, may prevent its movement altogether. This condition greatly increases the difficulty of diagnosis.

Respiratory displacement of gastric tumours was formerly denied, and the differential diagnosis between tumours of the stomach and those of the liver, omentum, etc., was based on that fact—a conclusion which, in the light of more modern investigations, has been found to be erroneous. We must distinguish in these cases between cancer of the lesser curvature on the one hand, and cancer of the pylorus and fundus on the other hand. In the former, marked displacement may occur on inspiration, whereas on expiration the tumour is fixed. On the contrary, practically no inspiratory displacement occurs in carcinoma of the pylorus and of the fundus, excepting when the tumour is intergrown with the liver.[22] Inflation of the stomach is frequently of assistance in determining whether or not the carcinoma belongs to the stomach; in other cases it remains uncertain for a long time. The factors most intimately concerned in distinguishing between these conditions are presented in the following table:—

Tumour of the Liver	Cancer of the Gall-bladder	Cancer of the Pancreas	Cancer of the Intestines (Cancer of the Transverse Colon and Cancer of the Duodenum	Tumours of the Omentum
The edge of the liver moves with respiration; icterus frequently develops as the tumour increases in growth.	Icterus is present, as a rule; symptoms of direct dyspepsia or of dilatation of the stomach are absent; cancer of the gall-bladder is scarcely movable.	The head of the pancreas may occasionally be felt to the right of the median line; intense icterus is present, as a rule; the growth is immovable.	Cancer of the transverse colon presents phenomena of obstruction and causes alterations in the stools (blood, mucus, etc.); icterus is frequently present in duodenal carcinoma when it involves the region of the mouth of the bile-duct; the tumour is only slightly or not at all movable; bile and pancreatic juice in gastric contents.	Tumours of the omentum do not descend on inspiration; ascites is frequently present; the nodules are less circumscribed than in gastric cancer, and generally of a secondary nature.

I have included in it, to a large extent, the carefully prepared data of v. Leube,[23] which he had based on an extensive experience. I have digressed from this author's statements only in as far as I cannot agree with him, according to numerous observations, that free hydrochloric acid in the gastric contents is a distinguishing characteristic in doubtful abdominal tumours. I have, for instance, noted the absence of free hydrochloric acid in many cases of malignant abdominal tumours which were certainly not gastric in origin (confirmed by autopsies). Aside from the true tumours of the stomach-wall, the following conditions may simulate malignant growths: *hypertrophy of the muscularis, perigastric indurations, foreign bodies* in the stomach cavity, hairy conglomeration ("pilobezoars"), tumours consisting of vegetable fibres ("phytobezoars"[24] and others ["shellac-ball"]*), adhesion of the stomach-wall to other sections of the intestinal canal. A malignant tumour may, however, be excluded in such cases by careful observation of all diagnostic factors, such as an absence of cachexia, glandular swelling, œdema, and ascites; the probable diagnosis of a bezoar was made for the first time by Schreiber.[25]

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[22] Compare also Part II, page 567 *et seq.*

[23] v. Leube, Spezielle Diagnose innerer Krankheiten, Leipzig, 1889, S. 250f.

Fæcal tumours in the transverse or ascending colon may undoubtedly simulate neoplasms, and have unquestionably led even experienced examiners to make mistakes. The latter are best avoided by following the first examination by a second one after the bowels have been purged, a procedure which I follow in all doubtful cases. *It is wise, in any case, not to declare the presence of an abdominal tumour after the first examination, in cases which are at all doubtful.* Since numerous sources of error (full colon, better relaxation of the abdominal walls, quiet and regular respiration, etc.), are excluded by repeated explorations, considerably greater certainty may thus be attained.

Palpation of the abdomen itself should always be accompanied by that of the peripheral lymphatics, which include, for our purpose, the *inguinal* glands, the left *supraclavicular* glands, and the *umbilical* glands. The first are sympathetically enlarged in the majority of the cases of carcinoma of the gastric wall; yet they are only of slight diagnostic significance, for they are frequently present in other conditions, which cause similar phenomena. Swelling of the supraclavicular and umbilical glands[26] is rarely present in my experience, and is, therefore, of only secondary importance as a diagnostic criterion.**

(c) Splash-Sound, Succussion-Sound (Clapotement, Bruit de Clapotage).

The splash-sound is elicited through short, tapping strokes of the finger-tips against the region of the stomach, while the patient is in the recumbent position. This sound may be obtained to a lesser or greater extent under normal circumstances even in the *healthy* (sometimes when the belly-wall is relaxed or moderately fat, also soon after the ingestion of *large quantities of liquids,* one to two litres); as a rule, in that case the lower limits of the splash-sound do not exceed the horizontal umbilical line. *Under no circumstances, however, can the splash-sound be elicited in a normal fasting stomach.*

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* [A cabinet-maker used to drink the varnish which was employed in his work; a tumour was diagnosed during life; on post-portem examination a ball of shellac, the size of a small orange, was found in the stomach. Another quite similar case was reported by Manasse, Berliner klin. Wochenschrift, 1895, P. 723.]

** For further consideration of this subject, see Part II, page 581.

[24] Schönborn, Archiv für klinische Chirurgie, 1883, Bd. 29, S. 609.

[25] J. Schreiber, Mittheilungen aus den Grenzgebieten der Medicin und Chirurgie, 1896, Bd. 1, Heft 5, S. 29 (see there interesting discussion on the diagnosis).

[26] Boas, Archiv für Verdauungskrankheiten, 1901, Bd. 7, S. 426.

In *pathological* conditions, the extent and the nature of the splash must be determined, and the time which has elapsed since the last ingestion of nourishment must be taken into consideration. With reference to the first, it may be stated that when the splash-sound in normally-situated stomachs extends far below the normal limits, that is, below the horizontal umbilical line, a pathological state is indicated, and especially so when the sound appears two or three hours after a small, and five or seven hours after a large meal. These relationships vary, however, when the stomach occupies an abnormally low position. In that case, as was shown by Elsner,[27] a loud splashing sound may appear over a large area and below the horizontal umbilical line in individuals whose stomachs perfectly suffice the usual demands of normal living and of the normal ingestion of food.

The splash-sound by itself is in no way indicative of a diseased state of the stomach, and even less so of insufficient functional activity of that organ. For that reason other supplemental methods must be employed. Motor insufficiency may be suspected only when a particular form of the splash-sound is present. This is the splash which appears upon gentle tapping with the finger, over a large extent from the epigastrium to the symphysis (the superficial splashing of Sahli). It is usually associated with a loud succussion-sound, which is similar to that heard when a half-filled barrel is shaken ("Tonnengeräusch").

Several authors, especially Stiller,[28] Kuttner,[29] Volland,[30] Schüle,[31] Pariser,[32] and others, pronounce this splash-sound as a sign of atony, that is, gastric relaxation, even before it has reached the stage of demonstrable motor insufficiency. Accordingly, the splash-sound points toward atony (on which stress is laid by Stiller and Pariser) and, to a certain extent, as was shown by Penzoldt and Dehio, to an early stage of motor insufficiency, that is, a mild grade of insufficiency.

It must be admitted that this deduction is possible, yet it has not been proven. The splash-sound may likewise occur in health, so that when gastric disturbances are accompanied by a splash-sound during or even after a meal, it does not follow that it is a characteristic symptom. These disturbances, as a rule, resemble nervous dyspepsia (of which, as is known, a large percentage of cases do not present the above-mentioned acoustic phenomenon) so closely that the splash-sound may be considered a purely accidental symptom, or perhaps the result of changes in nourishment. At any rate, I have repeatedly observed in a large number of cases of so-called atony, that a diet which spares motor activity of the stomach not only fails to cause improvement, but indeed calls forth an exacerbation of the symptoms; whereas, on the other hand, gavage (forced feeding) brings about a gradual betterment, although the splash-sound need not necessarily disappear. Thus we approach the observations of Stiller, who recognised in atony the manifestation of an *universal asthenia*, although we cannot agree on the term "congenital."

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[27] Elsner, Berliner klinische Wochenschrift, 1901, No. 16.

[28] Stiller, Berliner klinische Wochenschrift, 1901, No. 39 and 50.

[29] Kuttner, same, No. 50.

[30] Volland, same, No. 43.

[31] Schüle, Archiv für Verdauungskrankheiten, 1901, Bd. 7, Heft 4 u. 5.

[32] Pariser, Balneologenkongress, 1902.

The splash-sound must be distinguished from the so-called gurgling and succussion-sound, which arises when the stomach contains only air, and none, or very little liquid, and when the epigastric portion of the abdominal wall is markedly retracted and arched forward alternatingly on account of contraction of the abdominal musculature. The succussion-sound may also be elicited in such individuals by rapidly pressing the abdominal muscle upon the stomach-wall while the patient is in the recumbent position; the patients themselves can only produce it when in the erect posture. This succussion-sound is of no diagnostic importance.

Percussion of the Stomach.

The object of percussion of the stomach is to determine the position and size of that organ, as well as of abnormal new growths originating in it.

Technique of Percussion of the Stomach.

Percussion of the stomach, in comparison with that of the organs lying above the diaphragm, requires an extraordinarily subtle technique. Thus, if results are to be of any particular diagnostic use, it is necessary at times to note the finest differences between the percussion-tones. Therefore it is necessary to employ exclusively finger-percussion in determining the borders of the stomach, and especially so because the percussing finger, after some experience, controls the result through palpation. Finger-percussion must be performed lightly; in determining the lower boundary of the stomach, the percussion must be extremely delicate, whereas in outlining the portion of the fundus which is covered by the right lung, stronger percussion is required under certain circumstances.

In percussion of the stomach, best results are obtained when the patient lies on his back, for in that posture the abdominal musculature is most completely relaxed. The outlining of the stomach can be best carried on in this position; yet under some circumstances it is always of value to percuss while the body is in the erect posture, namely, when the stomach is full, because the liquids then flow forward, and thus facilitate the determination of the lower gastric boundary. It must not be forgotten, to be sure, that the results of percussion, when the body is held erect, are not inconsiderably altered by the contracted state of the gastric musculature, which, of itself, gives a slight dulness.

In other cases, it is of advantage in percussion to have the patient lying on his side, which causes the fluid ingesta of the stomach to fall upon the side on which he lies, whereby the zone which is dull in the posterior position, becomes tympanitic. It is of little importance at what point percussion of the stomach is first begun; thus it is immaterial whether one begins from above or below, in the mammillary or in the parasternal line.

In my opinion, it is best to begin by percussing the lower border, as it represents the most important boundary, and that along the continuation of the parasternal line from the symphysis upward. Next percuss from the fourth rib downward toward the anterior axillary line until a tympanitic tone is obtained. This border is then marked with a blue pencil, after which percussion is continued in a horizontal direction toward the right, until a dull tone is obtained, which is caused by the close relationship of the lower boundary of the liver to that of the heart (about the fifth intercostal space, between the parasternal and the mammillary line). If now we imagine the above-mentioned line extended to the costal border, the whole portion of the fundus which lies in the hypochondrium is practically outlined.

Unfortunately, in percussion of the stomach (see Figs. 13 and 14), the portion of that organ which lies back of the anterior thoracic wall or abdominal wall, cannot, as a rule, be determined by percussion alone; this holds true only under normal conditions. On the other hand, if, for instance, the stomach has descended *in toto,* then the lesser curvature, when the organ is full, may be percussed over its entire extent and outlined from the liver. The same result may be obtained occasionally in a stomach of normal size when that organ is markedly distended with air. (See page 105.)

In percussion of the stomach, the following boundaries are to be outlined:—

1. The lower boundary.
2. The upper boundary.
3. The right boundary.
4. The left boundary.

The determination of the lower or chief border of the stomach may be quite difficult on account of its close association with the transverse colon, which may give a similar, or almost similar, percussion-sound. This difficulty is increased in case both contain the same contents (that is, air in both, or air and solid substances, etc.). This brings us at once to the proper mode of procedure, it being essential to obtain *good differential percussion-sounds* between the two divisions of the intestines. If, for example, both the transverse colon and the stomach contain air, the latter organ may be filled with water, whereby, in place of the previously indistinguishable tone, a loud tympanitic, partially tympanitic, or even dull sound is obtained. If both the transverse colon and the stomach contain solid substances, the two organs cannot be outlined unless resort may be had to other auxiliary measures (inflation); even in such cases, however, a difference may be recognised between the marked dulness of the transverse colon and the tympanitic dulness of the stomach. As has already been stated, such cases, which are by no means common, require a second examination after the bowels have been emptied. If the colon contains liquid contents, they gravitate downward, as was noted by Pacanowski,[33] when the erect posture is assumed, and are replaced by a slightly tympanitic zone between the stomach and the intestines, which disappears again in the recumbent position. Ex-

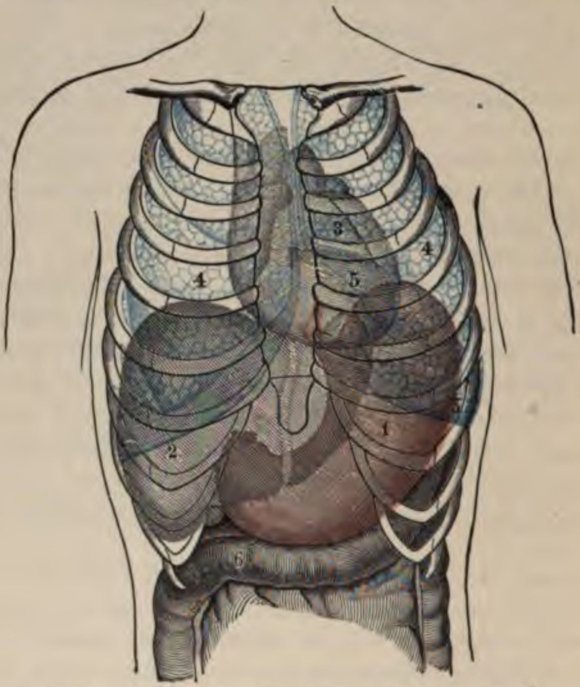

Fig. 13.—Position of the Stomach, Frontal Aspect.
1, Stomach. 2, Liver. 3, Heart. 4, Lungs. 5, Complemental pleural space. 6, Transverse colon. (From Eichhorst.)

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[33] Pacanowski, Deutsches Archiv für klinische Medicin, 1887, Bd. 40.

perience teaches us that in a certain small number of cases it is impossible, from a practical point of view, to outline the lower border.

Accordingly, we endeavour, as was already stated, to obtain artificial differentiating percussion-tones, usually by introducing air or liquids into the stomach. The latter method, which was first recommended by Piorry and later by Penzoldt,[34] depends upon the following observations:

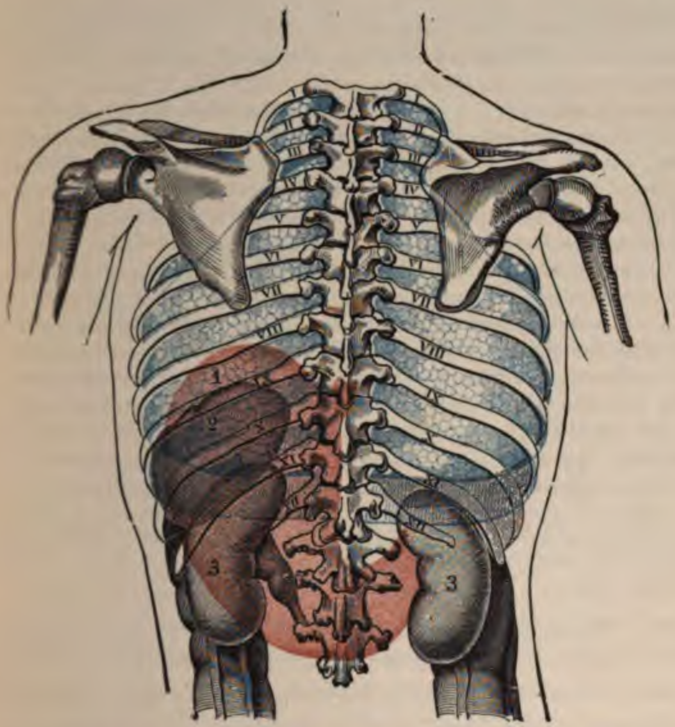

Fig. 14.—Position of the Stomach, Dorsal Aspect.
1, Stomach. 2, Spleen. 3, Kidneys. (From Eichhorst.)

When a litre of liquid is taken into a healthy fasting stomach, the dulness representing the greater curvature, when the patient is in the erect posture, is always found above the umbilicus. In an atonic condition, on the contrary, the dulness occurs more or less below the level of the umbilicus. This method resembles that proposed by Dehio.[35] He orders the patient at first to drink a quarter of a litre of water, and then outlines the position of the greater curvature, after which three more doses of a quarter of a litre of water are taken at short intervals.

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[34] Penzoldt, Die Magenerweiterung, Erlangen, 1877.

[35] Dehio, Verhandlungen des siebenten Kongresses für innere Medicin, 1888, S. 410.

The normal stomach, then, as was mentioned above, does not reach the level of the umbilicus while the patient is in the erect posture, whereas an atonic stomach descends far below it. Both methods have also the advantage of examination in the erect posture. It should be stated, furthermore, that no conclusion can be reached by the Penzoldt-Dehio methods as to the presence of a mechanical insufficiency, for diminution of elasticity and mechanical insufficiency can by no means be considered as identical.

Obrastzow[36] has made a detailed study of the lower border of the stomach by means of percussion and palpation. He divided the supraumbilical region, that is, the distance between the xyphoid process and the umbilicus, into three equal divisions, and found that the lower border of the stomach in men, as well as in women, lay in the lower supraumbilical third. In children under fifteen years of age, the lower gastric border rarely reached the horizontal umbilical line, whereas in persons above the age of fifty it is found frequently below the umbilicus; between these years the influence of age is not definite. After repeated pregnancies the lower border of the stomach is forced downwards. Likewise, all diseases accompanied by depression of the diaphragm, such as emphysema, pleuritis, and pneumothorax, cause the lower border to descend. Enlargement of the liver and spleen has a similar effect. The reverse holds true in all processes which press the diaphragm upwards, including, for example, the pregnant uterus and an extremely distended bladder.

Aside from the above-mentioned factors, the level of the lower gastric border is also influenced by nutrition; thus in persons of good constitution and nourishment, this border is found in the middle supraumbilical third, in mediocre nourishment it lies at the junction of the middle with the lower third, and in poorly-nourished subjects it is found near the level of the umbilicus.

The determination of the upper border (gastro-pulmonic) of the stomach likewise meets with not a few difficulties, for no distinguishable difference in the percussion-tone exists between the left lower pulmonic border and the highest portion of the gastric fundus. Yet, according to my observations, the moderate amount of air present, the size and the position of the stomach permit, to a certain extent, outlining this border. According to the conclusions of Pacanowski,[37] which coincide with the earlier investigations of Wagner,[38] the upper border lies:

In the *left parasternal line* at the lower edge of the fifth rib, or in the fifth intercostal space (rarely at the fourth or in the sixth intercostal space);

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[36] Obrastzow, Deutsches Archiv für klinische Medicin, 1888, Bd. 43, S. 417 bis 456.

[37] Pacanowski, *l. c.*, S. 342.

[38] Wagner, Inaug.-Dissert, Marburg, 1869.

In the *left mammillary line* in the fifth intercostal space as far as the sixth rib (occasionally also in the fourth intercostal space) or even at the seventh rib;

In the *anterior axillary line* at the lower border of the seventh or eighth rib, rarely below the sixth rib and never below the eighth rib.

The determination of this border is of but little value in comparison with that of the lower gastric border. It may, on the other hand, become of importance in left-sided pleuritic exudates, pneumonia, emphysema, and pneumothorax, in which the semi-lunar space of Traube (which lies, as is well-known, above the arch of the ribs, between the same, the edge of the liver, lung, and spleen, and accordingly corresponds to the fundus of the stomach) becomes more or less restricted in size. It follows, therefore, that the area of the fundus of the stomach may be smaller, even though in itself it is in no way changed in size and position.

The *right* border of the stomach is likewise difficult to determine and is of little diagnostic value (gastro-hepatic border). Pacanowski recognises (see above) a *lower* right gastric border, which lies to the right of the median line below the anterior edge of the liver, and an *upper* right gastric border, which is bounded by the left lobe of the liver. The upper portion, which lies at a point five centimetres from the median line, is alone determinable. The left gastric border (gastro-splenic border) can be determined only under favourable circumstances, because of the difficulty of outlining the spleen by percussion;* it, too, is only of secondary importance.

It is of interest to the diagnostician to determine the greatest *height and breadth of the stomach* by percussion, that is, the distance between the highest and lowest points as well as the furthest point to the right and that to the left at which percussion can elicit gastric tympany. Pacanowski (see above), who, after Wagner, presents the most accurate measurements in this respect, found that the greatest height is always less than the greatest breadth. Following are the measurements in the male and the female.

	Height	Breadth	Relation of height to breadth
Men	11 to 14 centimetres	21 centimetres	1 : 1.5 to 2
Women . . .	10 centimetres	18 centimetres	1 : 1.8 to 2

The outline of the stomach, on percussion, may become larger or smaller, depending upon various circumstances. It is *larger* in the physiologically large stomach, in gastroptosis, gastric ectasia, contraction of the left lobe of the liver, and finally in left-side pulmonic contraction.

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* [The percussion of the spleen becomes less difficult if, when the patient is examined, while in the recumbent position he is tilted slightly to the right side and his left arm is elevated till the hand reaches the top of the head.]

It is *smaller* than normal in enlargement of the left lobe of the liver, in which the gastro-hepatic border is displaced downward and to the left; also in left-sided pleurisy, rarely in pneumonia, left-sided emphysema, pneumothorax, splenic enlargement, and cardiac hypertrophy; in these cases the upper portion of the stomach (gastro-pulmonic border) is smaller in size. The percussion-outline of the stomach is also changed with the posture of the stomach, especially in the vertical position of the stomach, and also in the *situs inversus,* in which the cardia and fundus are moved to the right and the pylorus to the left.

Aside from determining the outline of the stomach by percussion, it is also important to percuss any tumours which might originate in the gastric walls. These tumours, which lie on an air-containing membranous bag, give, as a rule, modified tympanitic sounds on percussion, and are distinguished from hepatic and pancreatic tumours, which produce an absolutely dull tone. Yet, as was shown by v. Leube, a gastric tumour may also give a dull tone.

Auscultation of the Stomach.

Even though no characteristic significance can be attached to auscultation of the stomach, yet it is not altogether without diagnostic value. With reference to this subject, the following conditions are to be considered:

1. Auscultation of the Deglutition Sounds.

As we know, two sounds are heard during deglutition when auscultation is practised in the neighbourhood of the ensiform process or posteriorly in the region of the tenth dorsal vertebra. The first follows directly upon the determination of the swallowing act and gives the impression as if a liquid were forcibly injected through an air-containing space. Meltzer[39] has therefore designated it as "Durchspritzgeräusch" [squirting sound]. Ewald[40] calls it the "primary murmur." This is followed in from a few to about twelve seconds by a second sound, which is less clear and resounding and more like a "large bubble;" this is called the "secondary sound" by Ewald, and the "Durchpressgeräuch" [pressing sound] by Meltzer.

The primary sound, for reasons still unknown, may not occur, but the second sound is far more constant, although it, too, may occasionally fail. Under pathological conditions, that is, in constriction of the cardia, the normal appearance of the second sound will be considerably delayed, and its tone is also altered, because the fluid is gradually forced through in a jerky and gurgling manner. Under these circumstances this sound may not appear for fifty to seventy seconds and requires several seconds before it has completely disappeared. When the cardia is completely or almost completely obstructed, both the primary and secondary sounds may be wholly absent, this is not, however, absolutely pathognomonic, for, as was stated, the deglutition-murmurs may not be heard even when the cardia is intact. On the other hand, the presence of the first and second sounds within the normal time proves unconditionally the absence of œsophageal or cardiac stenosis.

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[39] Meltzer, Centralblatt für die medicinische Wissenschaften, 1883, No. 1.

[40] Ewald, Klinik der Verdauungskrankheiten, 1893, Theil 1, S. 64 u. f.

2. *Auscultation of the Gastric Murmurs.*

When the stomach is filled with air and liquids a splashing sound is heard under various circumstances when the patient changes his position. This murmur is also called the *succussion-sound,* and resembles the sound obtained when a cask containing water is slowly shaken. The latter is of the same diagnostic significance as the splash-sound. If the succussion-sound is so pronounced that it is heard over a large area, even upon the slightest change in position, or even in the fasting stomach, the presence of marked stagnation of the gastric contents is indicated.

The so-called gurgling and succussion-sound has already been mentioned (p. 86). The squirting, bubbling, singing sounds which are heard upon auscultation of the stomach are of no practical importance. According to my observations, sounds are occasionally distinguished which appear at certain intervals and which seem to be the acoustic effect of peristalsis and the action of the latter upon the chyme; these sounds possess more of a "lapping" character, as if a liquid were flowing rapidly along a wall. The remaining sounds owe their origin more to the development of air-bubbles, which arise even normally during the digestive act; in abnormal gas fermentation these may, however, develop to an exceptionally high degree. Murmurs of this type are never present in the fasting stomach. The above-described sounds are indicative of disturbances in digestion only when they are noted a long time after ingestion of food (five to seven hours after the principal meals, three to four hours after the smaller meals), and when they are particularly intense.

A few observers (Williams, Thorspeckler) claim to have heard in a few cases of rupture of the stomach a distinct sound like a sharp report. I have no personal experience to offer on this subject. Strümpell[41] calls attention to a sound which was widely heard and synchronous with respiration in a case of gastric dilatation. Laker[42] refers to a quasi bell-ringing sound in a patient suffering from gastric disease, which in his opinion was caused by the strong pulsation of the abdominal aorta or of the heart-beat against the near-by dilated stomach.

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[41] Strümpell, Berliner klinische Wochenschrift, 1879, No. 30.

[42] Laker, Wiener med. Presse, 1889, No. 43 u. 44.

[Benedict has described another form of sounds for diagnostic purposes to determine the degree of acidity in the gastric contents. He artificially made the sounds by introducing bicarbonate of soda into the stomach, which on contact with the acid causes the sound of effervescence.]

Auscultatory Percussion of the Stomach.

Laennec, the founder of auscultation, had already auscultated the sound which is heard when percussion is performed in the neighbourhood of a stethoscope placed over the pneumothorax cavity. He proved thereby that so long as one percusses over the pneumothorax a clear, resonant tone is heard which may occasionally be mistaken for a metallic character, but that this peculiar tone was no longer heard when the percussion reached the limits of the cavity. In this manner the extent of the cavity could be determined. Heubner[43] and Leichtenstern[44] have later called attention to the so-called rod-percussion, and recommended it for outlining contiguous organs. This form of percussion has been practically confined to outlining distended coils of intestines, and has never received general application. Auscultatory percussion has received greater attention in more recent times and has been energetically recommended by many authors (Henschen,[45] Bendersky,[46] Runeberg,[47] Bianchi,[48] Buch,[49] Pal,[50] and others). Bendersky ascribed the greatest importance, in auscultatory percussion, to the increased distinctness of the percussion-sound, whereas Henschen and Runeberg came to the conclusion that if practical and useful results are sought, one must desist from all attempts at determining the shades in the percussion-sound of the designated organs and confine oneself to outlining the auscultated organ. Henschen states that if in auscultating over a large air-containing cavity, percussion is practised at any other point over the same cavity, the percussion-sound will be transmitted to the auscultating ear with great intensity, provided the percussed point is not separated from the auscultated point by an intervening tissue of any kind. As soon as the area outside of the cavity is percussed, the sound becomes weak and distant, and loses the peculiar ringing tone and intensity which characterise it when one percusses over the cavity. Runeberg showed that the same principles hold true for cavities which are filled with liquid, and, indeed, for either air-containing or airless cavities, also for circumscribed growths.

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[43] Heubner, Archiv der Heilkunde, 1869, S. 326.

[44] Leichtenstern, Deutsche Klinik, 1873.

[45] Henschen, Upsala läkare sällsk. förhandling, 1887-88, Bd. 23, S. 420-424 (quoted from Buch).

[46] Bendersky, Wiener medicinische Wochenschrift, 1894, No. 36.

[47] Runeberg, Zeitschrift für klinische Medicin, 1901, Bd. 42, S. 81.

[48] Bianchi, Wiener medicinische Wochenschrift, 1898, No. 30 u. 31.

[49] Buch, Deutsche medicinische Wochenschrift, 1901, No. 38.

[50] Pal, Wiener medicinische Wochenschrift, 1902, No. 8.

"The chief object is the limitation of the percussion-phenomenon by the wall surrounding the auscultated organ, or, in case of very light percussion, by the boundaries of an intervening organ" (Runeberg).

The technique of the *transsonance method* consists in the use of the ordinary stethoscope, or of a flexible stethoscope. The funnel is applied to the organ to be examined with the left hand, which moves it about as may be desired. The transsonance note is produced by tapping or gently percussing with the tip of the index finger of the right hand. The boundaries as they are determined may be marked with the dermographic pencil. [The use of the double stethoscope (also phonendoscope) is of good service just for occasions of this kind.]

In this manner one may easily detect the boundaries between the uncovered portion of the liver and the edge of the lung, between the heart and liver, and between an exudate bordering on any of these organs; furthermore, even though it be more difficult, the boundary between the portion of the liver covered by the lung or heart, and the portion of the stomach covered by the liver can also be determined. Percussion of the spleen is also easily accomplished in this manner. The outline of the greater curvature is distinguished from the colon with great ease. This is performed by placing the stethoscope at a point which, by previous examination, was found to lie within the borders of the stomach. From this point percussion is performed downward, and in doing so, a tone is produced which, until the lower border is reached, has a loud and high tympanitic sound; this, however, becomes duller the nearer one approaches the limits of the gastric fundus. The transverse colon, which lies beneath this border, is outlined in a similar manner. I have convinced myself of the usefulness of the transsonance method in a large number of cases, and consider it of value especially to control the ordinary method of percussion. An advantage of the method is the ease with which it can be carried out.

Examination of the Stomach By Means of the Gastric Tube.

The employment of the stomach-tube is intended for the following diagnostic purposes:—

1. To determine the presence of œsophageal obstruction, especially of the cardia.
2. To determine the position and size of the stomach.
3. To remove the stomach-contents in order to study the digestive process in individual cases.

These subjects are discussed in detail as follows:

1. For this purpose the ordinary English sounds are employed, or the Nélaton tube, which is now generally used for the stomach. I agree with v. Leube[51] that the first examination of a stenosis should also be made with the Nélaton tube (Tube Faucher). Not until an obstruction has been localised is it permissible to cautiously employ the English sound. I do not fear slight hæmorrhages which result in this procedure, provided a sufficiently careful examination had been previously made, for they are never followed by unfavourable sequelæ. On the other hand, microscopical examination of bits of tissue which occasionally adhere to the sound may, under favourable circumstances, demonstrate the diagnosis of the disease which causes the stricture (for example, carcinoma of the œsophagus or of the cardia). I consider the black and so-called French sounds, which are composed of flexible hard rubber, as unsuitable, for it is impossible to properly sterilise them and they are easily broken.

2. v. Leube formerly employed the hard English sounds to determine the position and also the size of the stomach. It is occasionally possible, in that case, to feel the point of the sound through a relaxed abdominal wall, and by that means to locate the lower border of the stomach, which may also be done by palpation by way of the rectum. v. Leube[52] himself, however, has abandoned this diagnostically uncertain and dangerous method. A far simpler and less hazardous method of establishing the position of the stomach and, as I have shown, also of the greater curvature, consists in palpating, through the abdominal walls, a *soft* gastric tube which has been introduced into the stomach. Under those circumstances, in the majority of stomach diseases, the whole section of the sound which lies along the wall of the greater curvature can be located with the palpating hand. I have demonstrated in a large number of cases that the position of the pylorus may likewise be determined with certainty, or at any rate with great probability, by palpation of the tip of the tube.

In order to settle any dispute as to whether the tube really follows the direction of the greater curvature, I,[53] in connection with my former assistant Schmilinsky,[54] traced the route of the tube in an excised stomach which was joined to the œsophagus. The following observations were made: The tube passes at first along the descending limb of the greater curvature (Fig. 15, A). At this point, meeting its first resistance, it turns and glides along the greater curvature to the pylorus (B). Here the tip of the tube again meets an obstruction. If now the tube is pushed further in, it bends with its convexity toward the fundus; this curvature of the tube is at first slight, but it becomes more and more arched, until finally the tube lies to a more or less extent entirely along the wall of the lowermost portion of the fundus (C).

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[51] v. Leube, Spezielle Diagnose der inneren Krankheiten, 1889, S. 224.

[52] v. Leube, *l. c.*, S. 253.

[53] Boas, Centralbl. f. innere medicin, 1896, No. 6, und Deutsche Medicinal Zeitung, 1896, No. 22 (Lecture delivered before the Hufeland Society, in Berlin).

[54] S. A. Schmilinsky, Archiv für Verdauungskrankheiten, 1896, Bd. 2, Heft 2.

If done skilfully, this procedure may be performed in nearly all patients suffering from diseases of the stomach, and especially in those who are accustomed to the passage of the tube. I was successful, even in my early attempts, in more than 80 per cent. of all cases, and now the percentage is even greater. Jaworski,[55] who showed that tube-palpation was an important advancement in the physical examination of the stomach, states that it can be successfully performed in about one-fourth to one-third of all cases examined. It is probably not necessary to emphasise that the

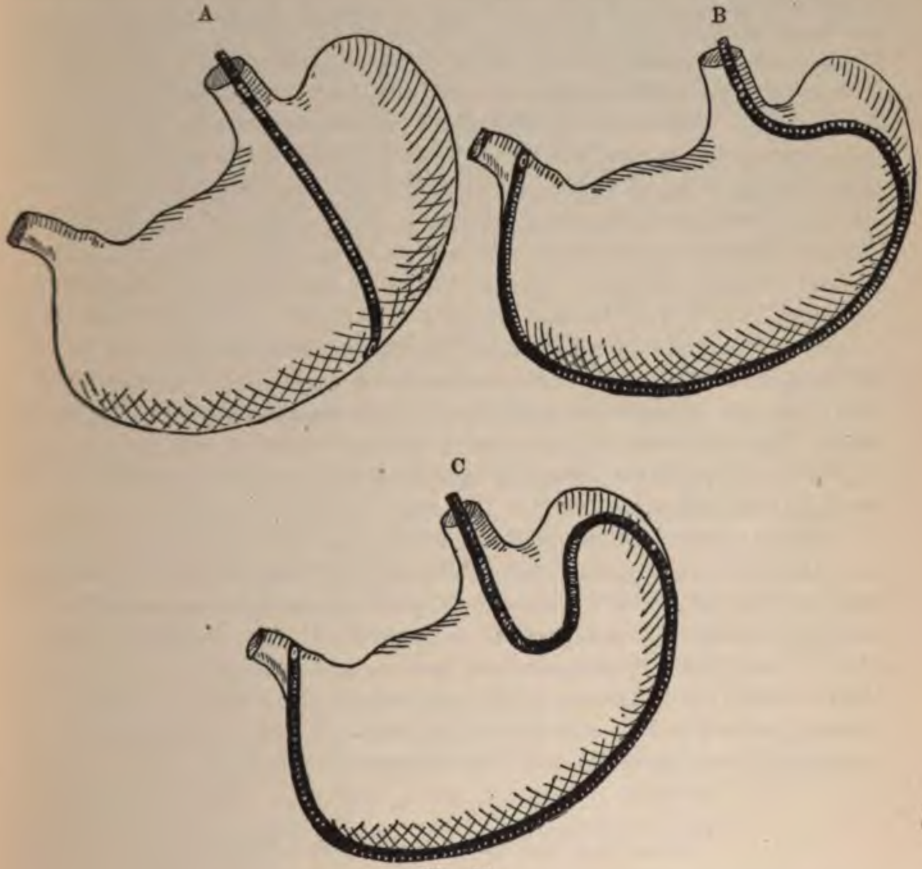

Fig. 15.

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[55] Jaworski, Wiener medicinische Presse, 1897, No. 51.

sound-palpation can only be practised successfully on emaciated individuals —which, to be sure, is the condition of the majority of patients suffering from stomach-trouble.

The following points are to be noted in the *technique* of sound-palpation: the examination can be executed most satisfactorily when the stomach is empty or only moderately full (one-half to one litre of liquid, patient being in recumbent position). In order to prevent outflow of gastric juice, it is wise to apply a clamp to the upper end of the tube. If one is not acquainted with the position and the size of the stomach, it is always preferable to employ a rather long tube (about 80 to 100 centimetres). Aside from the recumbent posture, the tube may occasionally be introduced in the erect position. In this manner it is possible to palpate either a large or small section of the tube which lies along the greater curvature. When any uncertainty exists as to whether the body in question is the tube or a contracted loop of intestines, it is well to direct the patient to slowly remove the tube. If then the resistance disappears, one may be convinced that it was the tube which was palpated. Tube-palpation may be employed not only for outlining the greater curvature, but also to better localise indefinite tumours (stomach, intestines, liver, omentum, kidneys), as well as doubtful painful areas (stomach, intestines, omentum, etc.). The soft tube may also be of service indirectly to determine the position and size of the stomach, by employing it to inflate or to transilluminate that organ.

3. The removal of the stomach-contents by means of the tube represents the most important and significant of our various diagnostic methods. Thus is obtained not only a clear insight into the chemical processes of the stomach, but also the condition of the motor activity. It offers, furthermore, a therapeutic advantage which has not been credited with the importance it deserves. By means of the information thus obtained we may, for instance, order a patient a diet which is not based upon theoretic reasoning, but upon the results of chemical and microscopical analysis.

Form and Quality of the Stomach-Tube.

A useful stomach-tube must, in my experience, fulfil the following requirements:—

1. It must be composed of an elastic, yet not too flexible nor too soft a material. The latter is especially likely to increase the difficulty of the introduction of the tube for the first time. Aside from the English Jacques patented tubes, other varieties of suitable (and far cheaper) stomach-tubes are purchasable in Germany [and America].

2. It must be at least 72 to 75 centimetres long and 6 millimetres thick (children require shorter and narrower tubes). Sometimes, as, for instance, in case of

descended or dilated stomach, the length of the tube must measure 80 to 100 centimetres.

3. Its lower end must have a blunt or a slight conical shape.

4. It should be supplied with but two lateral fenestræ, which are placed as near the end as possible. The diameters of the lateral openings should equal 1½ to 2 centimetres. Their edges must be absolutely smooth and blunt; torn edges may easily injure the gastric mucous membrane. Tubes having the so-called sunk openings are very useful.

5. I do not think it practical to have other smaller openings, as was recommended by Schütz and later by Ewald, for they interfere with absolute cleanliness, or at least make the latter more difficult, and furthermore possess no advantage.

Absolute antisepsis is of great importance to physicians who frequently analyse gastric contents or practise gastric lavage. This is especially true for the application of the tube in lues, cancer, and tuberculosis.

Physicians are recommended to employ new tubes in their private practice under all circumstances when gastric lavage is indicated. In the poor-practice and in institution work a separate tube should at least be kept for cases of cancer, lues, and tuberculosis. In other cases it is sufficient to boil them thoroughly in a solution of lysoform.

Technique for Introduction of Stomach-Tube.

Since the introduction of the elastic (Nélaton) tube into the diagnosis of stomach-diseases (Ewald, Oser), the application of tubes has made considerable advancement in regard to simplicity and the absence of danger. Fatal cases, during or after the passage of gastric sounds, as were not uncommonly observed formerly, when stiff sounds were employed, are now, since the use of the soft stomach-tube, together with the proper regard for contra-indications (page 103), rarely, if ever, seen.

With reference to the technique employed in the passage of the stomach-tube, attention should be called to several important points which, as I have observed, are the source of much difficulty, especially to beginners.

1. In order to determine the presence of any anomaly (chronic pharyngitis, tonsillitis, pharyngeal ulcers, syphilomata, etc.), which might interfere with the passage of the stomach-tube, that procedure should always be preceded by careful inspection of the mouth and pharynx. *At the same time, if a set of teeth is present which is not absolutely tight, it should be removed.*

2. Avoid the introduction of one or even more fingers when passing the tube (as was the custom when the soft sound was first employed), and instead urge the patient himself to swallow it. Simultaneous with the latter act of the patient, seek to pass slowly over the laryngo-pharyngeal opening, which is usually felt through the tube by a slight jolt. After this point is passed, by gently shoving, the tube will glide into the stomach, provided no obstruction is met in the œsophagus. The passage of the tube from the cardia into the stomach is likewise felt in rare cases by a slight jolt.

3. While passing the tube, request the patient to breathe deeply through his nose and at the same time to look directly at your eyes. So long as the latter is possible, all is in order.

4. The majority of the patients have a tendency to instinctively bend the head backwards during the passage of the tube. This makes breathing difficult and interferes with the circulation. Therefore direct the patients, before introducing the tube, *to bow the head forward.* [Tell the patient to sit upright and to bend the chin downward (soldier's position of head).]

5. On account of the awkwardness of the physician or great excitement of the patient, the tube may glide into the larynx instead of the pharynx. This mistake is immediately recognised by the cyanosis which develops, and the laryngeal respiration. It would be a grave error to fail, under these circumstances, to withdraw the tube immediately.

6. The pharynx needs to be anæsthetised only rarely, as in case of marked general hyperæsthesia, or in chronic pharyngitis, especially in tobacco-smokers. I resort, for this purpose, with advantage to thorough painting of the pharynx with 5 to 10 per cent. solution of cocaine, or 5 per cent. eucaine solution, or to repeated gargling with a 10 per cent. solution of potassium bromide shortly before passing the tube. [The gargling with a plain table-salt solution is frequently quite sufficient. To leave the tube quietly resting for a few seconds at the posterior pharyngeal wall, before further introduction, is often of great service.]

7. Of further importance is the dexterous removal of the stomach-tube after the gastric contents have been withdrawn. This is best done by compressing the upper end with two fingers of the right hand and the lower end with two fingers of the left hand. The contents which are in and on the tube are then allowed to run into a vessel held below it.

8. I likewise consider that the introduction of the fingers always increases the difficulty in the application of the stiff (English) sounds as well as of whale-bone sounds for the exploration and overcoming of œsophageal strictures. I guide the sound, holding it as a pen is held, as far as the posterior pharyngeal wall while the mouth is held as wide open as possible, and guide it over the epiglottis with a careful, pendulum-like motion. I have thus frequently experienced that the simplest and most natural method facilitates this procedure, which is so unpleasant in every case to both the patient and the physician. Patients who have learned to pass the sound themselves prove that the customary introduction of the finger is an unnecessary procedure. Why, then, should not a physician with a good will and a little practice succeed in doing so himself?

9. If an obstruction is found with a sound, its position is noted by marking [with nitrate of silver] the sound at a point corresponding to the

front row of teeth; upon removal of the tube, carefully examine it for any blood, pus, body-tissue, or remnants of food which might cling attached to it, and preserve them for further examination. The sound is again passed, and notice taken whether the obstruction is found at the same position as was marked; determine what the calibre of a sound is which can pass the obstruction, and whether a second obstruction exists, and finally whether a pouch is present. Occasionally this examination must be completed with the so-called English sound, yet in many cases (ulcerating carcinoma, etc.) the application of the Nélaton tube is the safest, and therefore the most preferable.

The following figures are of importance in locating the obstruction: The average length of the œsophagus in the adult is 25 centimetres; the distance from the incisor teeth to the cardia in adults is about 40 centimetres (in the new-born, about 17 centimetres); the distance from the incisor teeth to the bifurcation of the trachea equals about 21 to 22 centimetres.

Indications and Contra-indications for Examination of the Stomach with the Tube.

The employment of the tube is not indicated in every digestive, or even gastric, disturbance; on the contrary, we deem it directly contra-indicated in a large number of diseases. In general, it may be stated that the exploratory employment of the tube is only indicated in those cases in which it is impossible or difficult to make a diagnosis from the other symptoms. It is contra-indicated whenever the procedure is of danger to the patient, or when a diagnosis can be made from the other symptoms alone. We speak of a relative indication when the object of the application of the tube is to complete the diagnosis or to obtain information of value to the therapy.

The number of indications which are hard to tabulate can be more easily presented by noting the contra-indications.

The exploratory use of the stomach-tube is contra-indicated in the following conditions:—

A. In constitutional or local diseases in which the irritation associated with the passage of the tube may exacerbate the disease, or may even endanger the life.

These include:—

1. Heart-disease in the stage of failure of compensation, and angina pectoris, myocarditis, and advanced fatty heart.
2. Aneurism of the large arteries.
3. Recent hæmorrhages of any source whatever (lungs, stomach, kidneys, bladder, uterus, cerebral, hæmorrhagic infarcts, etc.).

4. Advanced stages of phthisis.
5. Advanced stages of pulmonary emphysema, with bronchial catarrh.
6. Apoplexy, partial or complete; cerebral hyperæmia, epilepsy.
7. Pregnancy.*
8. Presence of continuous or remittent fever.
9. High-grade cachexia.
10. Old age.

B. In gastric or intestinal diseases which can be diagnosed without the passage of the tube.

These include:—

1. Ulcer with very recent hæmatemesis or black stools.
2. Palpable gastric carcinoma, with emaciation, coffee-ground-like vomitus, and other classical signs of cancer.
3. Many gastric neuroses, in which the character of the disease is indicated by the other symptoms.
4. Acute gastric or intestinal catarrh, accompanied by fever.
5. Gastric mucous membrane which tends to bleed easily (slight capillary hæmorrhages, however, do not figure as contra-indications).
6. Secondary gastric affections which, without further examination, are known to be dependent upon other primary affections (phthisis, nephritis, diabetes, gall-stones, etc.).

C. During Menstruation. Tests for gastric secretion during menstruation may, according to the researches of Hans Elsner,[56] yield results which vary considerably from those obtained at other times. Changes in the motor function, however, could not be observed at that time.

While the above statements, on the whole, can hardly be contradicted, yet I wish to supplement a word or two as to my position against the application of the tube in gastric ulcer. I agree with Flade[57] that the employment of the tube is not justified either in suspected or in atypical cases of ulcer, for the gastric contents play no distinguishing rôle in either case. It is best to simply regard a doubtful case of ulcer as ulcer, and to treat it accordingly.

[While the application of the tube for diagnostic purposes in gastric ulcer may be superfluous, for therapeutic purposes, the tube may be introduced, especially if the neighbourhood of the cardia can be excluded as the seat of the ulcer, and I have frequently used the same with great benefit to the patient.]

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* The tube for the purpose of gastric lavage, as is well known, is occasionally employed with good success for the continuous vomiting of pregnancy. Otherwise I consider pregnancy as a contra-indication for the exploratory use of the sound.

[56] Hans Elsner, Archiv für Verdauungskrankheiten, 1899, Bd. 5, S. 467.

[57] Flade, Münchener medicin. Wochenschrift, 1901, No. 32 u. 33.

Inflation of the Stomach.

(Inflation with Carbonic Acid and Air.)

(a) The carbonic acid inflation of the stomach with effervescing mixtures, which was introduced into the diagnosis of gastric diseases by Frerichs and Mannkopf, and since then generally adopted in the practice of medicine, is intended to facilitate the study of the position and the size of the stomach. It is best performed by dissolving 4 or 5 grammes of tartaric acid in a half glass of water, which the patient is requested to drink; this is followed by an equal amount of sodium bicarbonate dissolved in a like quantity of water. Much larger doses are recommended by v. Ziemssen[58] (7.0 sodium bicarbonate and 6.0 tartaric acid for the male, and 6.0 sodium bicarbonate and 5.0 tartaric acid for the female stomach). Inflation with carbonic acid is not dangerous, but it is self-evident that *under no circumstances is it to be employed in round ulcer or when fresh adhesions between the stomach and intestines are suspected.*

[Dr. M. Behrend,[59] Philadelphia, reports three cases of death alleged to be due to inflation of the stomach with CO_2. The cases refer to three patients, namely: a woman aged sixty-eight years, a man aged seventy-three years, and a man aged fifty-two years. While the lives of these patients were, as Behrend says, "undoubtedly shortened by the inflation of the stomach with CO_2," the use of CO_2 for inflation of the stomach must not be condemned on account of these three unlucky occurrences. According to the histories of these three cases, inflation was, if not directly contra-indicated, at least *not* indicated. It is not evident from the histories what real purpose was to be gained through the inflation, while, on the other hand, it appears that other diagnostic methods for eliciting the real condition of the stomach were not employed. The third case is very interesting on account of the enormous dilatation of the œsophagus, and it is strange that the report does not attempt to give any cause for it; autopsy report does not give any stenosis of the cardiac portion. I shall very briefly mention one case.

Case II, report says: "June 28, 1901, œsophageal bougies pass easily." Hardly thirty-six hours later post-mortem examination shows: "At the junction of the œsophagus and stomach a hard mass may be felt externally. Beginning 5 centimetres from the cardiac orifice there is a mushroom-like tumour, soft in consistency, taking in entire diameter. This is continuous with a similar growth extending to the stomach at orifice, involving the entire circumference." Behrend's report of these three cases alone does not at all justify him to sum up as he does: "The practice of using these methods (the use of CO_2) in the office and dispensary should be condemned."]

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[58] H. v. Ziemssen, Klin. Vorträge, 1888, No. 12, S. 13.

[59] Behrend, The Medical News, December 19, 1903.

Agreeing with v. Ziemssen, I cannot share the apprehension of Pacanowski[60] in regard to abnormal distension of the stomach when that organ is otherwise intact.

The advantages of this procedure are unmistakable; the whole stomach is lifted forth from the abdominal cavity at one time, its form can be sharply outlined by the eye and the palpating fingers of the examiner, and indistinct or invisible or non-palpable tumours can now be observed.

To be sure, this condition of the stomach lasts only so long as CO_2 is being generated; as soon as the chemical process is completed, the carbonic acid escapes very rapidly, either upward or downward, and the stomach again collapses. This interferes with careful examinations, especially in complicated cases. The development of carbonic acid does not cause the stomach to be sharply outlined in every case, probably because the irritation of the carbonic acid excites peristalsis, which rapidly forces a portion of that substance into the intestines. In that case, as Kelling[62] also states, there is danger of mistaking a distended intestinal coil for the stomach.

(b) Inflation of the stomach by means of a double bulb. This consists in arming a tube with a double bulb (as applied to the spray apparatus), introducing the tube into the stomach, and then gradually inflating it while the patient is in the recumbent position. This method, first recommended by Runeberg,[63] and practised for a long time by Oser[64] and Ewald,[65] did not become useful until the introduction of the stomach-tube into general practice. The inflation of the stomach with air possesses obvious advantages over carbonic-acid method. These consist in the ability to regulate the amount of air as may be desired; thus it may be diminished (by aspiration or expression), and after it has escaped it may again be increased. Furthermore, the amount of air required is of diagnostic value. Thus, for instance, a relaxed, ectatic stomach requires a far greater amount of air for complete distension than such as retains its tone. The contour of the stomach is, furthermore, much sharper and distinct, so that, as a rule, the whole organ can be easily palpated in its division of the abdominal wall. On the other hand, distension of the stomach with the tube and double bulb is rarely successful at the first attempt; a certain practice, and suppression of the gagging reflexes, must precede it.

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[60] Pacanowski, Deutsches Archiv für klin. Medicin, 1887, Bd. 40.

[62] Kelling, Volkmann's Sammlung klinischer Vorträge, 1896, N. F., No. 44.

[63] Runeberg, Deutsches Archiv für klinische Medicin, Bd. 34, S. 460.

[64] Oser, Die Neurosen des Magens und ihre Behandlung, 1885, S. 10.

[65] Ewald, Klinik der Verdauungskrankheiten, Bd. 2, 3 Auft., S. 80.

Inflation of the stomach also requires great caution and careful observation of the patient during the procedure. The air must be allowed to escape as soon as the patient experiences any pressure or tension, and especially pain in the region of the stomach (which he can best indicate by a previously arranged signal). Also avoid sudden inflation, and inflate the stomach as slowly as possible [by slight but rapid compression of the bulb]. Owing to this precaution I have never observed any untoward results in the many ambulatory and private patients in whom I have practised gastric inflation.

In many cases it is commendable to blow only very small quantities of air into the stomach, for in this manner the contour of that organ becomes more prominent than if large quantities are employed. Furthermore, when the stomach is only slightly inflated its normal outline is better recognised than when it is distended to abnormal proportions.

The *contra-indications* to inflation of the stomach include those for the passage of the tube as well as those for inflation with carbonic acid, that is, gastric ulcer and adhesions of the stomach-walls, and perhaps also marked meteorism of the stomach and the intestines.

Bouveret,[66] and later Fürbringer,[67] recommended blowing air into the stomach directly with the mouth through a tube. In order to avoid the disturbances accompanying sudden swallowing and vomiting movements, Fürbringer and Krönig[68] recommend passing the tube only about half-way down the œsophagus instead of into the stomach, and then blowing air into it while in that position. The only advantage which I can attribute to this method of inflation of the stomach is the fact that the double bulb, which is not always at hand, can be dispensed with. On the other hand, this method is not so agreeable to the physician, because of the gagging and vomiting movements which have already been referred to. The possibility of drawing carcinomatous gastric contents into the mouth should also be considered. The recommendation of Spivak,[69] to let the patient himself inflate the stomach with his own mouth, seems more theoretical than practical. It may be of advantage to some patients to inflate the stomach through the œsophagus—concerning which I have had no experience—yet whether that method is preferable to that of Runeberg is indeed doubtful.

Occasionally, both inflation with carbonic acid, as well as that with air, does not cause the stomach to become distended nor its contour to become prominent, although such an effect is produced in the intestines. This condition was first described by Ebstein[70] as *incontinence of the pylorus*, and later similar observations were made by Stiller[71] and Wilkes.[72] I have had occasion, although very rarely, to confirm these observations. The stomach emptied itself abnormally rapidly in such cases, and I suspected that the insufficiency of the pylorus went hand in hand with the too rapid expulsion of the (undigested) contents into the intestines. However, on the other hand, I would like to state that in cases of very rapid expulsion of the gastric contents the stomach may nevertheless show a pylorus which closes very well. Kelling, as well as Rostoski,[73] has also called attention recently to the possibility of distending the colon by inflation of the stomach, and referred to the mistakes which might arise therefrom.

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[66] Bouveret, Traité des maladies de l'estomac, Paris, 1893.

[67] Fürbringer, Deutsche medicinische Wochenschrift, 1899, No. 40.

[68] Krönig, same, 1899, No. 44.

[69] Spivak, same, 1900, No. 23; [Philadelphia Medical Journal, February 3, 1901. I have used Spivak's method successfully at various times, and some patients seem to enjoy the method].

[70] Ebstein, Volkmann's Sammlung klinische Vorträge, No. 155.

Minkowski[74] recommended a combination of carbonic acid inflation of the stomach with distension of the large intestines by means of water. The abdominal tumours tend, under those circumstances, to turn into the direction *where the organ to which they belong is normally situated.* Minkowski has observed, with reference to the stomach, that tumours of the anterior wall and greater curvature, when the organ is distended, feel broader and less distinctly outlined, whereas neoplasmata of the lesser curvature tend to disappear entirely,* because the greater curvature is projected markedly forward. Tumours at the pylorus generally move to the right and downward. When the intestines are filled with water, all tumours of the stomach simply move downward.

Determination of the Position and the Capacity of the Stomach.

The determination of the capacity of the stomach depends upon examinations made partly on cadavers, and partly on living subjects, and the amount of water or air required to completely fill the gastric cavity is taken as a standard of measurement. The results obtained from filling the stomachs of cadavers showed such extraordinary variations that they can only be accounted for by differences in the conditions of the tests. Following are some of the figures taken from Ost[75]:—

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* This is, however, not always the case, for in several of my observations on cancer of the lesser curvature, the tumour, on inflation of that organ, moved somewhat backward, although it remained distinctly palpable.

[71] Stiller, Wiener medicin. Wochenschrift, 1879, No. 4 u. 5.

[72] Wilkes, Ueber die Insufficienz des Pylorus, Inaug.-Diss., Bonn, 1885.

[73] Rostoski, Münchener medicinische Wochenschrift, 1900, No. 40.

[74] Minkowski, Berliner klinische Wochenschrift, 1888, No. 31.

[75] Ost, Gesammelte Abhandlungen aus der medicin. Klinik in Dorpat, edited by Prof. Unverricht, Wiesbaden, 1893.

Ewald	250 to 1680	cubic centimetres.
Luschka	1500 to 1200	" "
Schüren	2430	" "
Beneke	3000	" "
Brinton	3130	" "
Soemmering	2500 to 5500	" "
Henle	2500 to 5500	" "

Ewald[76] applied the results obtained by him on the cadaver directly to the living being, which for obvious reasons, is not permissible.

Kussmaul[77] found the capacity of the stomach in a phthisical patient to be 2600 cubic centimetres; he declared the gastric capacity of 2500 cubic centimetres in a shoemaker forty-eight years old, who was 140 centimetres tall, was decidedly too large. Quincke[78] found the same capacity in a sixteen-years-old person who was healthy, excepting for a gastric fistula. Jaworski,[79] who also experimented by filling the stomach with water, states that, as a matter of fact, one can never be sure whether or not the stomach is completely filled, for the weight of the water acts only upon the lower portion of the stomach walls and causes them to distend, whereas little or no effect is produced upon the upper portion of the stomach. It is furthermore questionable whether the living stomach can be completely filled up to the cardia; the sensation of weight, the pain, and the tendency to vomiting probably make this impossible. Jaworski, on these grounds, considers it preferable to fill the stomach with air rather than with water.

In fact, much depends in this connection, according to my experience, upon the momentary condition of the stomach, the quietude of the patient, his familiarity with the tube, and many other factors, all of which influence more or less the accuracy of the procedure. The capacity of a stomach can only be considered pathological when it is capable of holding an excessive amount of fluid, that is, somewhat over 3000 cubic centimetres; no positive conclusions whatever can be made upon a smaller capacity. Aside from simply filling the stomach with water, or with air, which is by far the more accurate, a number of more or less ingenious methods have been devised for the purpose of determining the position and the capacity of the stomach; these possess, however, in general no practical value. We mention them below only in order to be complete:—

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[76] Ewald, Klinik der Verdauungskrankheiten, 3 Auft, S. 86.

[77] Kussmaul, Volkmann's Sammlung klinische Vorträge, No. 181.

[78] Quincke, Archiv für experim. Pathologie u. Pharmakologie, 1889, Bd. 25.

[79] Jaworski, Deutsches Archiv für klinische Medicin, 1884, Bd. 35.

(a) The Method of O. Rosenbach.[80]

This method is intended not only to learn the position of the stomach, but also to determine the mechanical power of the organ. It depends upon the fact that the level of the fluid in the stomach, which may be determined by observing the rise and fall of the surface, suffices as a criterion for judging the resistance of the gastric fundus against the distending fluid.

The determination of the surface of the fluid will afford the possibility of obtaining absolute figures for the fall and rise of the same. For this purpose the tube which has been introduced into the stomach is connected with a rubber bulb. By placing the ear against the abdominal wall, a large bubbling, moist, and often metallic rattling will be heard as soon as the fenestra of the tube is immersed into the fluid; this is followed by a resounding and distinct splash. If, on withdrawing the tube, it is no longer immersed in the fluid, one will hear either no sound whatever, or only the hissing murmur due to the inflation of air.

The method is performed in the individual case as follows: Determine whether the stomach is empty.* Next, in case of adults, 50 to 100 cubic centimetres (one-half of that amount is sufficient in children) are introduced into the stomach through the tube, and while the bulb is being compressed auscultation is performed. The difference in the length of the tube and that which exists when the râles disappear represents the measurement of the depth of the fluid in the stomach. Accordingly, the length of the portion of the tube which is withdrawn equals about the diameter of the lumen of the stomach. If insufficiency of the gastric musculature exists, the level of the fluid in the stomach, when that organ is being filled, rises much slower on account of the atonic musculature. The slower the level of the fluid rises, the shorter is naturally the difference between the length of the tube when the splashing is heard and that when it disappears. That quantity of fluid which, when introduced into an empty stomach, causes no elevation, or even a sinking, of the level of the stomach, represents the extreme limits of the mechanical function of the stomach. In this manner an idea is obtained of the quantity of liquid to be overcome by the stomach, which offers valuable therapeutic aid.

(b) The Method of Neubauer.[81]

This method depends upon the well-known principle of two communicating tubes. The level of the water in the funnel of a stomach-syphon is at the same height as that in the stomach, provided air can enter the stomach along the side of the tube. This procedure is best performed with a double-current tube. O. Rosenbach (*l.c.*), simultaneously with Neubauer, proved this method to be simple and practical, yet it is only rarely employed at the present time.

(c) Method of Fleischer.[82]

Fleischer employs as a manometer a small U-shaped tube filled with liquid, which is attached to the free end of an ordinary rubber tube. The tube is then introduced into the stomach, and as soon as the fenestra at its end is submerged below the level of the liquid, the air in the tube becomes compressed and the fluid in the U-shaped tube is forced downward. In this manner the time when the tip of the tube enters the fluid, or, in other words, the level of the liquid in the stomach, may be exactly measured by the graduated manometer. Of course the effect of the respiratory movements must be excluded; this is best accomplished by cutting another small opening into the tube below the upper one. If now this opening is closed with the finger and the tube is pushed down to the level of the fluid, while the patient holds his breath, the immersion of the fenestra of the tube into the fluid will be followed by a change in the height of the fluid in the manometer, uninfluenced by respiration. This procedure has likewise gained no foothold in practice.

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* The expression method is probably the best suited for this purpose.

[80] O. Rosenbach, Volkmann's Sammlung klinischer Vorträge, 1878, No. 153.

[81] J. W. Neubauer, Prager medicin. Wochenschrift, 1878, No. 74.

[82] Quoted from v. Leube, Die Magensonde, 1879, S. 74.

(d) Method of Schreiber.[83]

The method of Schreiber consists in introducing into the stomach an œsophageal tube, to the lower end of which a small, thin rubber bag is attached. By inflating the bag, he sought to obtain conclusions as to the size and relative position of the organ. Schreiber considered it especially useful to determine the situation of tumours of the stomach and its neighbouring organs, which are outlined with difficulty.

(e) Method of Queirolo.[84]

In this method a small bladder is attached to the lower end of a tube which is inserted into the stomach. The oral end of the tube is connected with two rubber tubes, to one of which a registering pen is attached, and to the other a small stopcock. The small bladder is then inflated to a moderate size and the abdomen gently percussed; as soon as the percussing finger strikes the stomach the pen responds by making a stroke, but no response is obtained when structures outside of the stomach are being percussed. The subsequent tests of the Queirolo method by Edel and Volhardt[85] showed that no special advantage was offered by this procedure over the ordinary carbonic acid inflation, and, on the other hand, it is more complicated and requires more practice. According to these investigators it failed altogether when the stomach contained no food or air.

(f) Method of Jaworski.[86]

Jaworski fills the stomach with air, by means of a somewhat complicated apparatus, until the patient experiences a feeling of tension and pain in th҇ hypochondrium. The apparatus consists (see Fig. 16) of an elevated and, preferable, graduated bottle, which possesses a capacity of at least six litres. This is connected with a second Wulffian flask which contains about half to one litre of water, and which is supplied with three openings. One of the latter communicates with *A*, the second contains a manometer tube, and the third is connected with the gastric tube. Releasing the clamp at *b* permits the stomach to become filled with air. In order to ascertain the gastric capacity, the fasting stomach is first emptied as thoroughly as possible of gas and liquids by means of aspiration, after which air is allowed to enter the stomach in the above-mentioned way *until the feeling of tension is experienced* by the patient; the clamp *b* is then closed. The amount of water removed from *A*, or the amount added to the contents of *H*, represents the capacity of the stomach.

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[83] J. Schreiber, Deutsches Archiv für klinische Medicin, 1877, Bd. 19, S. 616; see also Archiv für Verdauungskrankheiten, 1898, Bd. 3, S, 221.

[84] Queirolo, Verhandlungen des 19. Kongresses für innere Medicin, 1900, S. 412.

[85] Edel and Volhardt, Deutsche medicinische Wochenschrift, 1900, No. 35.

[86] Jaworski, Deutsches Archiv für klinische Medicin, 1884, Bd. 35, S. 83.

(g) Methods of Kelling,[87] Unverricht, and Ost.[88]

Kelling and Ost, independently of each other, constructed an apparatus according to the same principles as the above-mentioned method but with greater regard to practical indications. By means of it the amount of air pumped into the stomach is determined by a simple calculation.

The apparatus of Kelling (Fig. 17) consists of a double bulb, a stomach-tube, an ordinary pail (basin, vessel), and a measuring cylinder holding two and one-half to three litres, which is graduated into divisions of 20 cubic centimetres; a simple manometer, consisting of a partly-filled U-shaped glass tube, supplied with a paper scale, is also needed. These various parts are then joined together in the manner shown in the above figure, with the assistance of strong gas-tubing, two T-shaped glass tubes, and a curved glass tube which tapers into a point at one end. Two clamps are then applied as shown in the above figure. The cylinder is then completely filled with water and inverted. Next the tip of the pointed tube is pushed as high above the level of water as possible, into the air-space which exists at the summit of the column of water within the cylinder (see illustration).

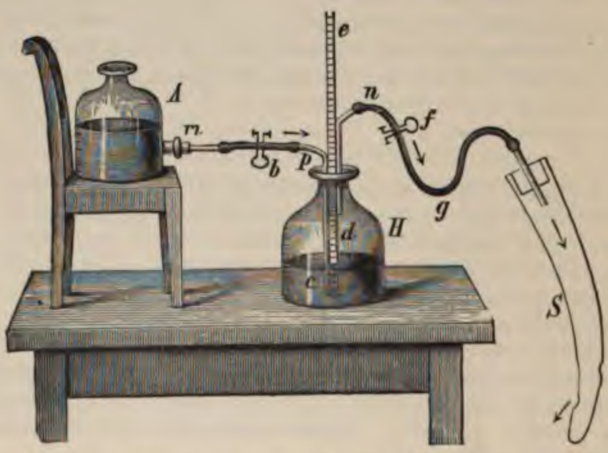

Fig. 16.—Jaworski's Apparatus for Estimating the Capacity of the Stomach.

This is done so as to keep the stomach as free of air as possible. The capacity of the stomach is determined in the following manner: The patient, who has been fasting in the morning, ingests a piece of wheat bread and two glasses of water; an hour later the stomach-tube is introduced and the stomach emptied of its contents by expression or by lavage. As soon as the stomach is emptied as much as possible, the gastric tube is connected with the upper T-shaped tube and the clamp nearest the measuring cylinder is applied. Then, while the patient sits in a chair or lies upon a sofa, the stomach is inflated by means of the double bulb.* If any of the air escapes along the side of the tube from the cardia or through a temporarily patent pylorus into the intestines, it must be replaced by more air.

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[87] Kelling, Deutsche medicinische Wochenschrift, 1892, No. 51 u. 52; Volkmann's Sammlung klinischer Vorträge, 1896, N. F. No. 44.

[88] Ost, *l. c.*

If the limits of the inflation have been reached, the clamp is applied to the double bulb and the one near the measuring cylinder is released. The air then escapes spontaneously from the stomach under water. If air no longer escapes, request the patient to press, or the air may also be assisted to escape by means of careful pressure upon the epigastrium. After the total quantity of air which had been introduced into the stomach is collected in the measuring cylinder, we must still calculate the amount of space which it occupied in the stomach. For all practical purposes, and without making any essential mistake in doing so, this calculation consists in adding 8 per cent. to the amount of air registered in the measuring cylinder, if the test is performed slowly, and 7 per cent. if done rapidly. If it is desired to avoid all calculation and directly employ the values registered, a mistake of 10 per cent. at the most is made, which would have to be added to the amount of the volume found.

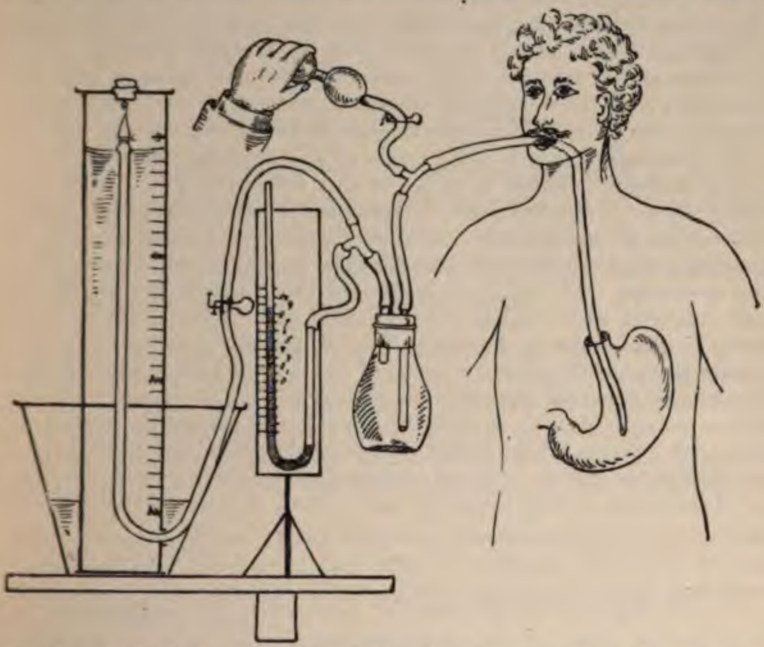

Fig. 17.—Kelling's Apparatus for Estimating the Capacity of the Stomach.

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* According to Kelling, it is essential to inflate the stomach as quickly as possible and likewise to suck the air out rapidly and energetically.

Kelling has proposed various formulæ for the accurate calculation of the capacity of the stomach, but it would involve too lengthy a discussion to be considered here. Kelling found in the above manner that the gastric capacity for air varies with the individual, but that on an average it was 1000 cubic centimetres when 20 centimetres water-pressure was employed. The pressure is higher in diseases accompanied by thickening of the gastric walls. The capacity of the stomach for water is, on the average, higher, the medium capacity being about 1500 cubic centimetres.

The method of Ost resembles the procedure just described. By means of a peculiarly-constructed pump 150 cubic centimetres of air can be introduced into the stomach at a time. Aside from this pump, an Hg-manometer for determining the intragastric pressure is also needed, as well as an apparatus for sucking the fluid from the stomach (measuring apparatus), and, finally, a stomach-tube. The measuring apparatus, whose base is furnished with an air-tight stopcock, is immersed by its lower open end into the water contained in a small basin, so that when the air is aspirated from the stopcock at its summit, the water rises from the basin into the cylinder. The pump, the manometer, the measuring jar and the tube are now joined to a convenient apparatus by means of a glass tube possessing four limbs. The first of the latter is joined, by means of a piece of rubber tubing, with the pump, and that with the ventilating tube through which the air is expelled. The second limb is joined to the stomach-tube. A small flask, with a double perforated stopper, is interpolated into the connecting piece, in order to prevent the entrance of vomitus into the apparatus. The third limb is attached to the manometer, and the last one is finally connected with a rubber tube, which is inserted into the open end of the measuring cylinder, where it is securely fastened, submerged in the water; this tube must, however, be tightly closed by means of a clamp when air is being pumped into the stomach. This method showed that a smaller amount of air was always pumped out of than into the stomach, and that the quantity of air lost in this manner was variable. Ost investigated the origin of this phenomenon, and found ***that the air escapes through the pylorus into the intestines, on account of which the determination of the capacity of the stomach by means of air is associated with unvaoidable errors.***

Criticisms of the Methods for Estimating the Capacity of the Stomach.

We must admit, even though we disregard the sources of error in the different methods, which in part are unavoidable, that the estimation of the capacity of the stomach should be taken in account for clinical purposes only with great caution; it is true, the estimation of the capacity of the stomach often offers a measurement of its mechanical power, but not always, as the loss of elasticity of the gastric walls is by no means proportionate to that of the expulsive force. The best example of this fact is demonstrated by cancer of the stomach, in which condition the dynamic strength suffers markedly, even in the early stages, whereas the loss of elasticity is comparatively slight, and indeed no loss whatever may occur. The most satisfactory methods are undoubtedly those devised by Jaworski and Kelling.

Pfaundler[89] compared these tests in the stomachs of children and obtained results which coincided with each other fairly well. The figures obtained by Pfaundler were, however, comparatively small, and he thought, therefore, that a certain amount of air is already present in the stomach before the filling test (Jaworski) is performed, and that when emptying the stomach, a like volume of air remains behind. Pfaundler was unable to confirm by his investigations the statements of Ost, namely, that air can escape either into the intestines or the œsophagus.

Pfaundler devised a method of his own (for children's stomachs), which excluded the above sources of error. Since this procedure has not hitherto been made applicable to the adult stomach, we will not describe it here.

Gastrodiaphany and Gastroscopy.

1. *Gastrodiaphany (Gastrodiaphanoscopy).*

Even though transillumination or illumination of the human stomach is not yet applicable to general practice, these methods offer much of interest, and point toward the road which, when improvement and simplification have taken place, will have to be followed.

The oldest method of diaphanoscopy was devised by Cazenave as early as 1845. In order to view the inner urethral walls, he employed a funnel-shaped metallic tube, which was introduced into the urethra by means of a mandrin. The light, which was reflected from a lamp and made more powerful by means of a collecting lens, was directed against the lower surface of the penis; in this manner it was possible to plainly see the inner wall of the urethra in spite of the thickness of the tissue.

Czermak and Gerhardt (1860) sought to transilluminate the larynx with sun- and gas-light, which likewise was done later by Störk and Voltolini. Fonssagrives reported in the same year on the transillumination of the cavities of the human body by means of Geissler's tubes.

Julius Bruck, a dentist of Breslau, deserves credit as being the first (1867) to have employed galvanic light for transillumination. He used an instrument similar to the vaginal speculum in exploring the rectum in the male and the vagina in the female; this appliance was connected with a Middeldorpf battery, the current of which brought a platinum coil to white heat and thereby transilluminated neighbouring portions of the bladder. Milliot[90] about the same time made the attempt, although only in animals, to transilluminate portions of the abdominal cavity by means of a light generated within a glass tube which had been introduced into the stomach or colon; the light is obtained from two thin platinum wires placed within the glass tube, which are connected with the electrodes of a battery. In 1868, a similar device was conceived by Dr. Lazarowicz (Charkow).

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[89] Pfaundler, Ueber Magenkapazitat und Gastrektasie im Kindesalter, Biblioth. medic., 1898, Abth. D,[1] Heft 5.

[90] Milliot, quoted from Schmidt's Jahrbuchern, Bd. 136, S. 143.

In more recent times, M. Einhorn,[91] of New York, prompted by the researches of Voltolini on electrical transillumination of the larynx, undertook to perform transillumination of the stomach (gastrodiaphany) by means of a simple procedure. He employed a Nélaton tube, to the lower end of which was attached an Edison hard-glass incandescent globe containing carbon filaments. This lamp was inserted in a metal attachment, which was connected with conducting wires; these ran through the tube to a battery. A current-interrupter was placed a short distance from the tube. The latter may be introduced as easily as an ordinary tube. Before beginning the test, the patient is given one or two glasses of water, after which the tube is lubricated with a little glycerine, and then guided into the stomach. Somewhat later, Heryng and Reichmann[92] proceeded in a similar manner, excepting that their apparatus is supplied with a continuous current of water; further investigations have proven this accessory to be superfluous. Einhorn's apparatus (Fig. 18), because of its convenience and the ease with which it can be introduced into the stomach, is most suitable for the purpose. The best source of light is a Hirschmann's accumulator [or some American accumulator]; when it is not obtainable, a strong galvanic battery will also serve the purpose. Heryng and Reichmann, and after them Einhorn, Renvers,[93] Pariser,[94] Boas, Kuttner and Jacobsohn,[95] more recently Martius[96] and Meltzing,[97] Schwartz,[98] Meinert,[99] Kelling,[100] Epstein,[101] Langerhans,[102] van der Weijde,[103] Schäffer,[104] Oppler,[105] Starck,[106] and others have all interested themselves in the application of gastrodiaphany and published their results. Before referring to their conclusions, we wish first to describe in a few words the technique of the procedure.

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[91] M. Einhorn, New York Medical Journal, November, 1889, and Berliner klinische Wochenschrift, 1892, No. 51.

[92] Heryng and Reichmann, Therap. Monatshefte, Marz, 1892.

[93] Renvers, Verein für innere Medicin, 4 April, 1892.

[94] Pariser, Berliner klinische Wochenschrift, 1892, No. 32.

[95] Kuttner and Jacobsohn, Berliner klinische Wochenschrift, 1893, No. 39.

[96] Martius, Gesellschaft deutscher Naturforscher und Aerzte in Wien, 1894; Centralblatt für innere Medicin, 1895, No. 49.

[97] Meltzing, Zeitschrift für klinische Medicin, 1895, Bd. 27, Heft 3 u. 4.

[98] Schwartz, Beiträge zur klinischen Chirurgie, 1895, Bd. 14, Heft 3.

[99] Meinert, Centralblatt für innere Medicin, 1895, No. 44; 1896, No. 12 u. 13.

[100] Kelling, Volkmann's Sammlung klinischer Vorträge, 1896, N. F. No. 44.

[101] Epstein, Jahrbuch für Kinderheilkunde, 1896, Bd. 41, N. F. Heft 3 u. 4, S. 328.

[102] Langerhans, Wiener medicin. Blätter, 1895, No. 45.

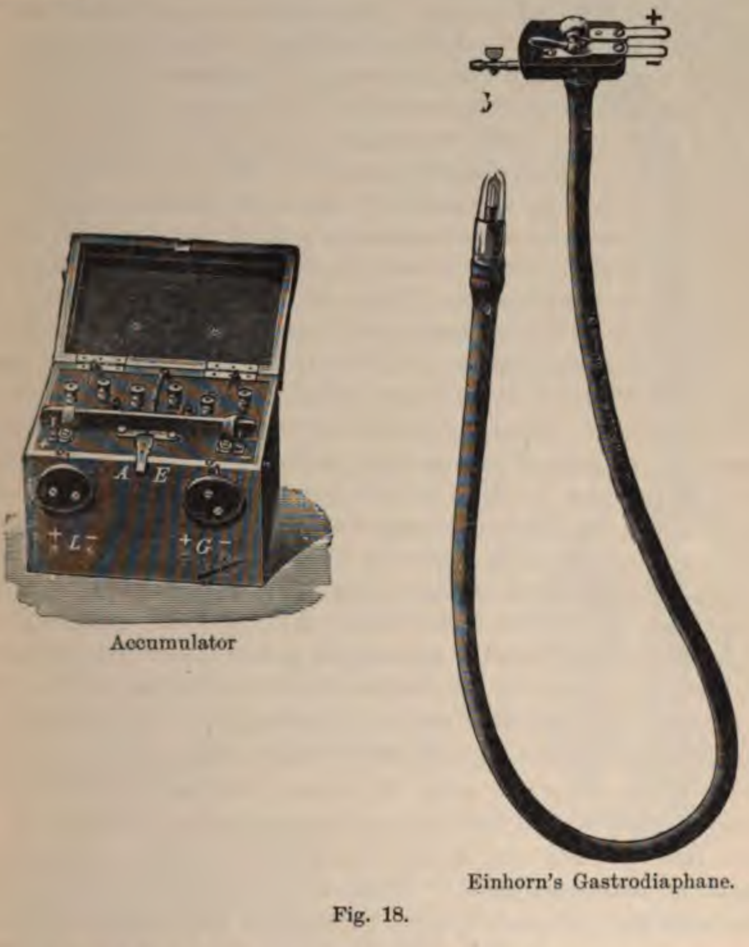
[103] van der Weijde, Nederl. Tijdschrift voor Geneeskunde, 1895, Deel II, No. 12.

[104] Schäffer, Inaug.-Diss., Bonn, 1896.

[105] Oppler, Archiv für Verdauungskrankheiten, 1897, Bd. 3, S. 334.

[106] Starck, Volkmann's Sammlung klin. Vorträge, 1898, N. F. 217.

Before beginning transillumination, it is absolutely necessary that the stomach contain no food, for otherwise no picture can be produced. Transillumination may therefore be practised on a fasting stomach, or, which is preferable, at other times after vigourous lavage of that organ. It is also recommended that the intestines and bladder be emptied before proceeding with the test.



Accumulator

Einhorn's Gastrodiaphane.

Fig. 18.

The patient then drinks one-half to one litre of water, or that quantity is introduced into the stomach through the tube. Kuttner and Jacobsohn employ a tube similar to the device proposed by Heryng and Reichmann, which is attached to the upper end of the gastrodiaphane (see Fig. 18*b*), to permit the inflow and outflow of the water; transillumination is best performed while the patient is in the erect position and in as dark a room as possible, although it may be done in the daytime* in a moderately dark room, provided strong lamps are employed. After the test has been completed, the water may again be removed by expression or syphonage.

Transillumination of the empty stomach has been rightly abandoned by the majority of authors. According to the position and contents of the stomach, variously-shaped pictures are obtained by transillumination, in which the curvatures of the stomach in particular, and occasionally also the gastric fundus, are completely reproduced. It is noteworthy, as was first shown by Renvers, and confirmed later by Kuttner, Kelling, and Meinert, that the transverse colon, which borders on the stomach, is to a greater or lesser extent also transparent to this light; this is represented by a crescent-shaped figure, which lies at the lower portion of the picture. The latter is obscured by the overlying liver, or by tumours attached to the anterior abdominal wall, or, finally, by intestinal coils containing fæcal matter. Shadows are furthermore cast by the abdominal musculature and the superficial veins of the skin.

Observations as to the practical utilisation of this procedure still differ from each other considerably. Einhorn attributes to it great practical importance and employs it for the following purposes:—

1. To rapidly recognise dilatation of the stomach. 2. To positively determine the presence of gastroptosis. 3. To detect tumours of the anterior wall of the stomach by their lack of translucency. Kuttner and Jacobsohn agree with the latter conclusion, but they do not regard the first-mentioned two conditions to be proven by the transilluminated figure; on the other hand, they attribute another diagnostic advantage to the picture thus obtained, namely, that the transilluminated figure shows respiratory movement in gastrectasia, which is not seen in gastroptosis. This is explained by the fact that in ectasia the fundus of the stomach lies in close contact with the diaphragm, which is not true in gastroptosis. This statement was contradicted on suitable grounds by Meltzing, Langerhans, and Kelling, whose views, on the whole, agree with mine. Perhaps the phenomenon of Kuttner-Jacobsohn holds true for complete gastroptosis, although the existence of that condition may be also determined by simpler methods, as for example, by inflation with air. Moreover, this method is of no value in determining the difference between gastroptosis and ectasia as we regard them in modern times, for these two anomalies present no contrasts. We agree wholly with Kelling[107] when he states that it is as incorrect

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* In my experience, a dark room is not necessary; in order to screen against too strong sun- or lamp-light, a so-called Spanish wall [screen] may suffice, or some similar contrivance.

[107] Kelling, *l.c.*, S. 503.

to ask whether gastroptosis or ectasia exists, as it is to ask whether gastroptosis or cancer is present.

No especial advantage can be conceded to transillumination, even though we acknowledge the value which is attributed to it by several authors. It was possible to establish the diagnosis of gastroptosis with certainty long before the gastrodiaphanoscope was discovered. Likewise transillumination of the stomach can protect us as little against error in the diagnosis of ectasia as any other physical method, for it is unable to express the state of the dynamic power of the stomach. Inasmuch as this condition can be determined with certainty by exploration with the stomach-tube, I cannot see what advantage is offered by the gastrodiaphane over the earlier and comparatively simpler methods. Finally, with reference to the diagnostic value of the diaphanoscope in carcinoma of the walls of the stomach, in tumours and displacement of the spleen (Kelling, Kuttner), in distinguishing between tumours of the gall-bladder and the pylorus, as well as in the recognition of phantom tumours (Kuttner[108]), it may be stated that this method plays a corroborative rôle. Taking all in all, the high expectations with which this procedure was originally received have not been fulfilled, and it is unnecessary, at least for the purposes of daily practice. The specialist, on the other hand, whose function it is to try all of the available methods in doubtful cases, may occasionally have use for transillumination.

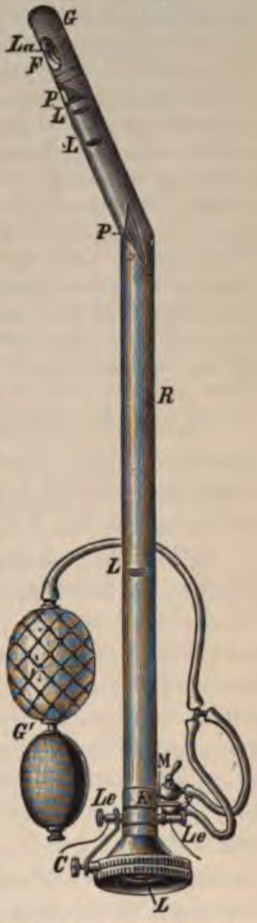

Fig. 19.—Leiter's Gastroscope.

2. *Gastroscopy.*

Gastroscopy has hitherto been found of only slight practical use, but it might undoubtedly play as important a rôle in the future, provided it is simplified, as cystoscopy has done in the diagnosis of diseases of the bladder.

The first gastroscope, that constructed by Leiter in Vienna (see Fig. 19), consists of a metal tube *R*, 57 centimetres long and 16 millimetres wide, which is bent at one end into an obtuse angle; it is supplied at one end with a conical-shaped and slightly-rounded cap *G*, which contains a window of rock-crystal plate *F* for the transmission of the rays of light from the lamp *La*. The conducting wires end at the ocular end of the instrument, in the battery screws *Le*. The contact contrivance *C* serves to open and close the instrument. The optical appliance is so arranged that the rays of light of the illuminated part of the gastric mucous membrane are at first guided to the prism *P*, which lies closest to the source of light, and then to two objective lenses of a terrestrial telescope, which lies within the tube; next they pass the prism *P*, located at the angle of the tube, enter the collecting lense at the upper portion of the instrument, which reverses the picture and at the same time projects it forward so that it can be directly observed through the ocular *L*. The terrestrial telescope is movable, so that it is possible to view a larger surface of the gastric mucous membrane. An air-bulb (*G'*) is attached to the apparatus, by means of which it is readily possible to force air into the stomach, in order to smooth out its folds. The air can be allowed to escape from the stomach through the little tube *v*. The apparatus is also furnished with an afferent and efferent water-pipe, which supplies water for the protection of the gastric mucous membrane against excessive heat. The gastroscope just described can only be employed to view the pylorus; in order to view the fundus and the other portions of the stomach, a second instrument is required, which differs from the former only by the fact that the window and the receiving prism are located on the posterior or lower side of the beak.

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[108] L. Kuttner, Berliner klin. Wochenschrift, 1896, No. 38.

Oser and v. Mikulicz[109] found that by means of the above-described apparatus, under favourable circumstances, single portions of the mucous membrane of the stomach may be illuminated, so that they can be plainly visible in both the healthy and the sick.

v. Mikulicz, particularly, was able to report a number of cases of cancer of the stomach, which he diagnosed by means of the gastroscope, also two cases of gastric ulcer, which yielded decidedly clear pictures. Of particular interest is the gastroscopic symptom discovered by v. Mikulicz in cancer of the pylorus, namely: whereas the healthy pylorus appears on gastroscopic examination as a rather long fissure, or a triangular, oval, or crescent opening, which is always surrounded by a circle of thick and markedly red folds of mucous membrane, v. Mikulicz found that in gastric cancer these folds were either altogether obliterated, or that only very superficial folds existed.

He explains this appearance by the fact that the organ, which was previously folded and movable, became converted by the carcinomatous infiltration into a rigid tube. v. Mikulicz also observed decided vasomotor changes in the region of the pylorus, consisting of anæmic, hyperæmic, and cyanotic areas, as well as bluish-coloured alterations in the submucous veins, which are likewise referable to circulatory changes following carcinomatous infiltration. According to v. Mikulicz, conclusions can be drawn as to the constancy of these symptoms and their diagnostic significance only after long observation.

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[109] v. Mikulicz, Wiener medicin. Presse, 1881, No. 45; Wiener medicin. Wochenschrift, 1883, No. 23 and 24.

In more recent times gastroscopic examinations have again been resorted to, especially by Rosenheim[110] and Kelling.[111]

Rosenheim's gastroscope is a strong, straight, metallic instrument, 68 centimetres long and 12 millimetres wide, which consists of three tubes which slide into each other (Fig. 20, *a*, *b*, *c*.). The inner tube (Fig. *c*) contains the optical apparatus, while the middle one (*a*) which ends in a rubber tip, to favour introduction, contains at *L* a little incandescent lamp, which is covered by a small glass window. Above this opening is a second one, *B*, in which, after the optical apparatus has been introduced, the prisms are located and where they can be moved about. The tube conveys both the contact wires as well as tubes for the constant current of water and for inflating the stomach with air. The third and outermost tube (*b*) is drawn over the last-mentioned, on which it may be rotated. It is furnished with a centimetre scale at its upper end and an opening at *E* corresponding to that at *B* (Fig. 20*a*), which is intended, by rotating to 180°, to cover the opening containing the prism and to protect the latter from being soiled upon introduction. The current is conducted in the customary manner, by means of contact pieces and Hirschmann's cables. The current of water is conducted by means of an irrigator, which is placed on a suitable stand. The source of light is the accumulator which is commonly employed.

According to Rosenheim the following rules must be observed before the application of the gastroscope:—

1. Make a functional test of the stomach, that is, determine whether the stomach is empty in the morning. If so, no special preparation is necessary; but if stagnation exists, the organ must be thoroughly washed out the evening before the test. Rosenheim, as a rule, introduces a 50-centimetre-long œsophageal tube before instituting gastroscopy, in order to remove any gastric contents which might be present.

2. The position, form, and size of the stomach are to be determined by the proper methods, especially the greater curvature and the position of the pylorus.

3. It is absolutely necessary to determine by means of a 70-centimetre-long hollow steel sound, whether the passage from the teeth to the greater curvature is free and can be passed without difficulty in the recumbent posture. An insurmountable obstruction is not rarely found at the diaphragm, due to spasm or abdominal curvature of the œsophagus. In such and similar cases, gastroscopy cannot be performed. After these preparations, the patient, while fasting, is examined on one of the following days. The gastroscope is introduced while the patient is on an operating table, with his

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[110] Rosenheim, Berliner klin. Wochenschrift, 1896, No. 13-15.

[111] Kelling, Archiv für Verdauungskrankheiten, Bd. 2, Heft 3 u. 4; Münchener medicin. Wochenschrift, 1897, No. 34, u. 1898, No. 49.

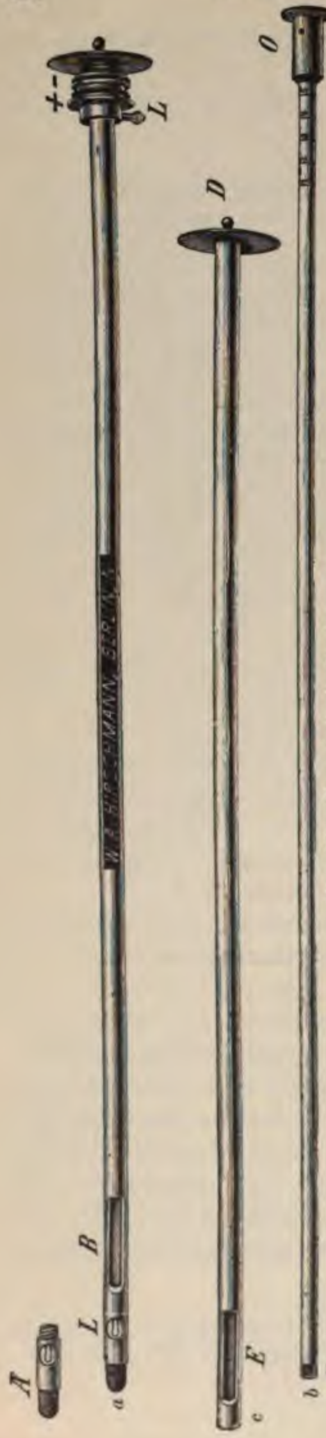

Fig. 20.—Rosenheim's Gastroscope.

head lowered and the hips elevated. It would entail too long a discussion to describe the various rules and precautions to be observed in performing gastroscopy, and even then the skill could not be developed which follows personal practice (it is better to practise first on the cadaver). It is possible, by means of the gastroscope, to view only the pylorus or the pyloric area, and perhaps also a segment of the lesser curvature which borders on the pylorus. Portions of the greater curvature, the posterior wall, the neighbourhood of the cardia, especially the portion which borders on the lesser curvature, cannot be seen. Finally, a "supplementary gastroscope" without the water-cooling attachment is described by Rosenheim, which is much thinner than the above-mentioned apparatus and is therefore more easily introduced. It possesses, however, the disadvantage of being heated too rapidly through the continuation of the current for the electric lamp, on account of which the examination can be continued for only a few seconds. Hence it is more useful for general reconnoitering than for the accurate determination of a questionable gastric disease.

Rosenheim already limited the application of the gastroscope, on good grounds, in his first publication, to such cases as have defied hitherto successful examinations with other methods. It was his chief endeavour to employ it in establishing the early diagnosis of cancer of the stomach. Gastroscopy was decidedly advanced by the instrument of Kelling (see Fig. 21).

The chief part of the apparatus consists of a jointed tube, which, from its bent portion to its head, measures 36 centimetres. A smooth and undivided metallic tube is attached to the joint at its head, the object of which is to permit a long section of the apparatus to be easily rotated and moved to and fro between the teeth. The greatest diameter of this tube equals 14 millimetres, which thickness is employed in its entire length.

The joints are smooth 2-centimetre-long cylinders, the greatest diameter of which equals 13 millimetres; the walls are 2 millimetres thick. Each joint is fitted with an extension to the right and to the left, equalling one-half the thickness of the walls of the tube, which connects with the surface of the preceding joint. These extensions are so fitted to the joint-surface by means of rivets that a hinge arrangement is formed. The heads of the rivets are filed down so that the points of their insertion are perfectly smooth both within and without. The extremities of the joints are filed off on the anterior surface; they are filed down horizontally on the posterior surface, so that when extended the joints lock with each other. A groove is filed posteriorly on the outer side of the joints, along which a wire passes. The wire is prevented from slipping out of the groove by a little bridge, which is soldered to each joint. The whole jointed tube is encased in a rubber tube. The principle of extension and flexion is the same as in the human finger; imagine the same as hollow. The extensor tendon is represented by the wire.

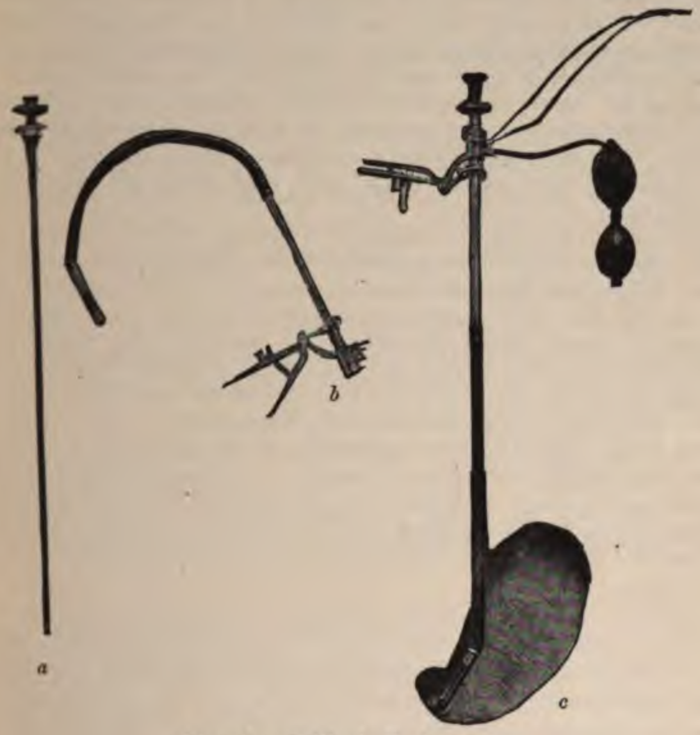

Fig. 21.—Kelling's Gastroscope.

The wire is drawn by a lever arrangement. The last joint is attached at a fixed angle to a tube fitted with a rounded cap, which represents the beak of the instrument. This beak measures about 10 centimetres in length (see Fig. *b*). The apparatus, bent to conform with the body-curves, is now introduced so far that the angle formed by the union of the beak with the jointed tube lies below the cardia. The apparatus is now straightened by compressing the handle, and fixed in that position by inserting a peg. The cap is then found to point toward the anterior wall of the stomach, with sufficient room to permit free movement.

The beak consists of two outer tubes. The first one, which is attached to the last joint by means of two hinges, extends about 3½ centimetres from the angle of the instrument. A second tube is attached to this, which contains the prism and the incandescent lamp. This tube can be rotated, while the former is naturally fixed. In order that the tube carrying the prism and the incandescent lamp can be easily rotated from without, it is screwed to an inner tube. The latter extends throughout the entire beak. After the apparatus has been rigidly fixed in the correct angle, a solid metallic tube is inserted into the jointed tube (see Fig. 21*a*). This tube reaches to the angle of the original tube. It is rounded, and is inserted by means of two niches in a cylinder which is situated at the angle, and which carries a cogwheel on the other side. The teeth of this wheel fit into those of the tube of the beak. The tube, which can be rotated within the beak, is firmly attached to the very lowest portion of the beak, which carries the prism and the incandescent lamp.

The stomach is inflated by means of a stopcock to which a double bulb is attached. The air travels through the whole apparatus, passes the incandescent lamp, and escapes into the stomach at the tip of the beak. This tip is furnished with a small valve, which prevents the entrance of mucus and water into the apparatus. A rotary disk is situated at the mouthpiece, to which a knob is fastened. This knob marks the position of the ocular prism. The tube is always introduced in such a manner that the knob lies in the same plane as the prism. When the tube is rotated, the prism and the knob conform to each other in movement, and we are thus able at all times to know the position of the prism in the stomach (Fig. 21*c*). The incandescent lamp is a so-called cold lamp, which can be kept illuminated as long as desired. The refraction of the light and the optical apparatus are very ingeniously constructed. It is, however, impossible here to enter into a detailed discussion of this subject, and therefore I refer the reader to Kelling's works.

According to Kelling, this apparatus is introduced with great ease. Nevertheless, its manipulation requires skilful technique and familiarity with the large number of mechanical details, practice on the cadaver and on dogs on whom œsophagotomy has been performed; accordingly, gastroscopy represents to a certain extent a specialty in itself. To these objections must be added those offered by the laity to uncomfortable methods of examination. Objections are still met with occasionally even to the passing of the simple stomach-tube. Finally, another great difficulty is found in the fact that this method is naturally not available in those cases in which we are most in need of it, namely, in establishing an early diagnosis of cancer of the stomach. For in my wide experience on this subject, patients rarely come under medical observation during the stage when an early diagnosis is to be made, for the simple reason that the patients have no complaints whatever at that time. However, even though slight symptoms exist, it is hardly probable that a patient will forthwith submit himself to a gastroposcopic examination because he is subject to a feeling of slight pressure, eructation, or loss of appetite. It remains, then, for the instrument to be employed in other doubtful cases (ulcer or cancer, hypertrophic pyloric stenoses, vague neuroses). In this respect, Kelling has contributed a series of fine observations and favourable operative results on these cases, which are undoubtedly encouraging.

Appendix.

The Application of the Röntgen Rays in the Diagnosis of Diseases of the Stomach.

The epoch-making discovery of Röntgen, the importance of which to surgery and certain branches of internal medicine (heart, blood-vessels, lungs, gout, etc.) cannot be denied, has also attracted the attention of the investigators to its employment in diseases of the abdominal organs. The Röntgen rays are undoubtedly also of great use occasionally in this respect, especially when metallic foreign bodies have been swallowed, which can thus be located with great certainty. Péan[112] and Raw[113] were thus able to locate coins which had been swallowed and had lodged in the cervical portions of the œsophagus. White[114] was able, on the evidence of a skiagram, to remove by operation a small metallic star from the stomach. Miller and Reid[115] found a swallowed dental set, containing six teeth, at the level of the sixth and seventh intercostal spaces. Pöch[116] obtained a photograph of a boy who had swallowed a so-called bread-mark (thin, coin-like piece of tin). By means of the fluoroscope one was able to observe the descent of the foreign body in the intestines from day to day.

The attempt has also been frequently made to make the size and position of the stomach and intestines visible; this was done either by inflation (Rosenfeld, Bade), or by introducing substances which cast shadows when exposed to the Röntgen rays, especially by the suspension of bismuth (W. Becher), and finally also, according to Wegele,[117] by introducing a metal spiral into the stomach. The first-mentioned method has hitherto only succeeded in producing vaguely-defined pictures of the position of the stomach, but E. Lindemann[118] has succeeded in obtaining excellent skiagrams by means of the last method, and there can be no doubt but that it is possible in this manner to determine at least the greater curvature, and probably also the fundus. Strauss,[119] and, independently of him, Levy-Dorn and myself,[120] proposed a procedure in which capsules filled with metallic bismuth are swallowed, in order to locate the greater curvature. In this manner the movements of the capsule can also be observed on the fluorescent screen, thus revealing the motor-activity of the stomach. Similar experiments with bismuth had already been previously performed on animals by Balthasard and Roux.

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[112] Péan, La semaine médicale, 1896, S. 494.

[113] Raw, British Medical Journal, 1896, S. 1678.

[114] White, Univ. Med. Magaz., Bd. 8, S. 9.

[115] Miller and Reid, quoted by Levy-Dorn, Deutsche medicinische Wochenschrift, 1897, No. 8.

[116] Pöch, Münchener medicinische Wochenschrift, 1897, No. 8.

[117] Wegele, Deutsche medicinische Wochenschrift, 1896, No. 18.

None of these methods has obtained a secure foothold in practice, obviously because their usefulness bears no proportion to the complicated and expensive apparatus which is needed.

The diagnosis of gastric and intestinal tumours by means of the Röntgen rays has heretofore been practically negative; and the prospects of more favourable results with the improvement of the technique are also very poor. Pöch[121] claims to have isolated single intestinal coils from each other, and indeed even to have seen the valvular folds, which, on the other hand, is doubted by Wullstein.[122]

[In April, 1905, Dr. F. L. Feldstein made some skiagrams for me in a case of a woman, sixty-one years of age, which showed very well the size, form, position, and motor function of a dropped stomach. In the last few years reports on the use of Röntgen rays in the diagnosis of diseases of the stomach have become more frequent and they seem to show that the x-rays are not to be neglected in the armamentary of the methods for diagnosis in the gastro-enteric affections. Besides the well-known experiments of Cannon[123] and Tousey,[124] it was Rieder,[125] in München, and G. E. Pfahler,[126] of Philadelphia, who in the course of their studies made beautiful and most instructive skiagrams for demonstration of position, size, form, and motility of the stomach and intestines. It is a great pleasure to me to express my thanks to my friend, Dr. Pfahler, for his kindness in furnishing me the four photographs which are published in this book for the first time. (See Plates II, III, IV, V.)]

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[118] Lindemann, Deutsche medicinische Wochenschrift, 1897, No. 17.

[119] Strauss, Deutsche medicinische Wochenschrift, 1896, No. 24, Vereinsbeilage.

[120] Boas and Levy-Dorn, Zur Diagnostik von Magen- und Darmkrankheiten mittels Röntgenstrahlen, Deutsche medicinische Wochenschrift, 1898, No. 2.

[121] Pöch, *l.c.*

[122] Wullstein, Berliner klinische Wochenschrift, 1857, No. 16.

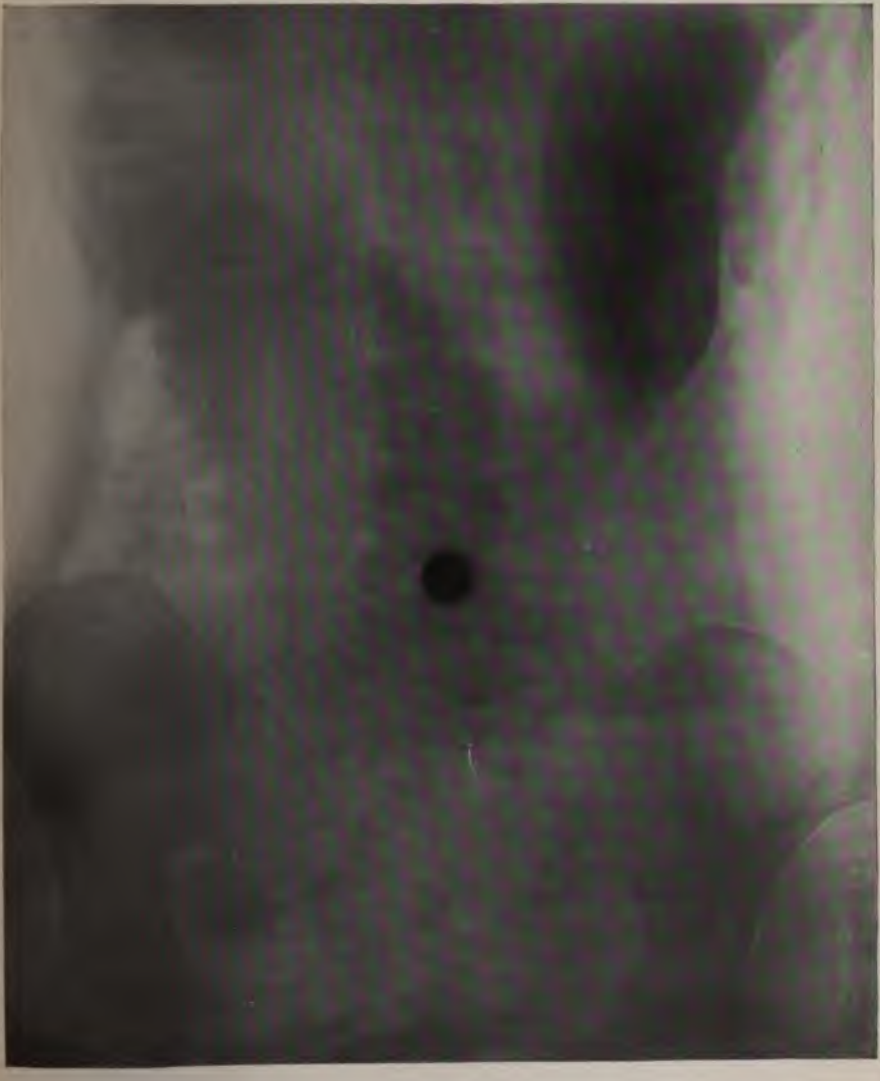
[123] Cannon, Journal of the American Medical Association, March 21, 1903.

[124] Tousey, New York Medical Journal, May 21, 1904.

[125] Rieder, Fortschritte auf dem Gebiete der Röntgenstrahlen, February 14, 1905.

[126] Pfahler, The Archives of Physiological Therapy, Boston, July, 1905.

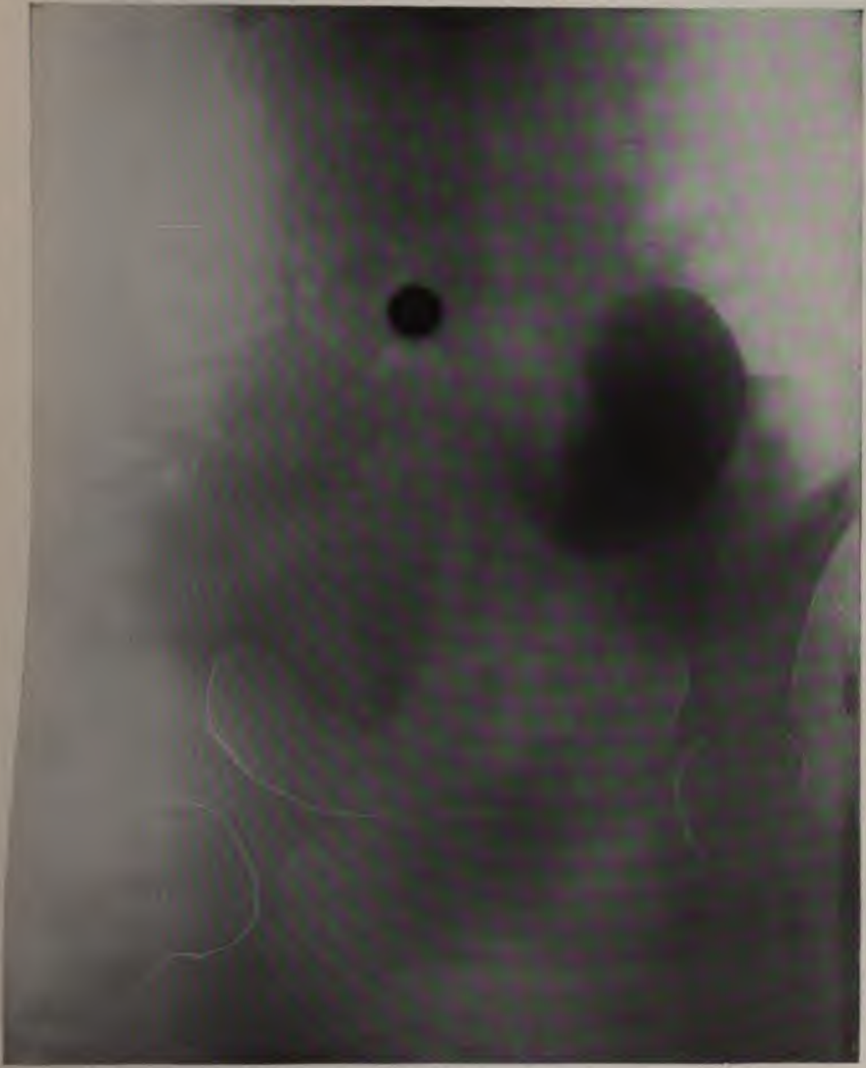
PLATE II



A case of gastroptosis. Examined in the prone position, just after taking an ounce of bismuth subnitrate and a pint of milk. Notice a cent on umbilicus; the stomach lying in a vertical position, entirely to the left of the median line, and the food just beginning to pass through the pylorus. The light area at the right side of the abdomen indicates the cæcum and the ascending colon.

(Service of Dr. W. E. Ashton. Skiagram by Dr. G. E. Pfahler.)

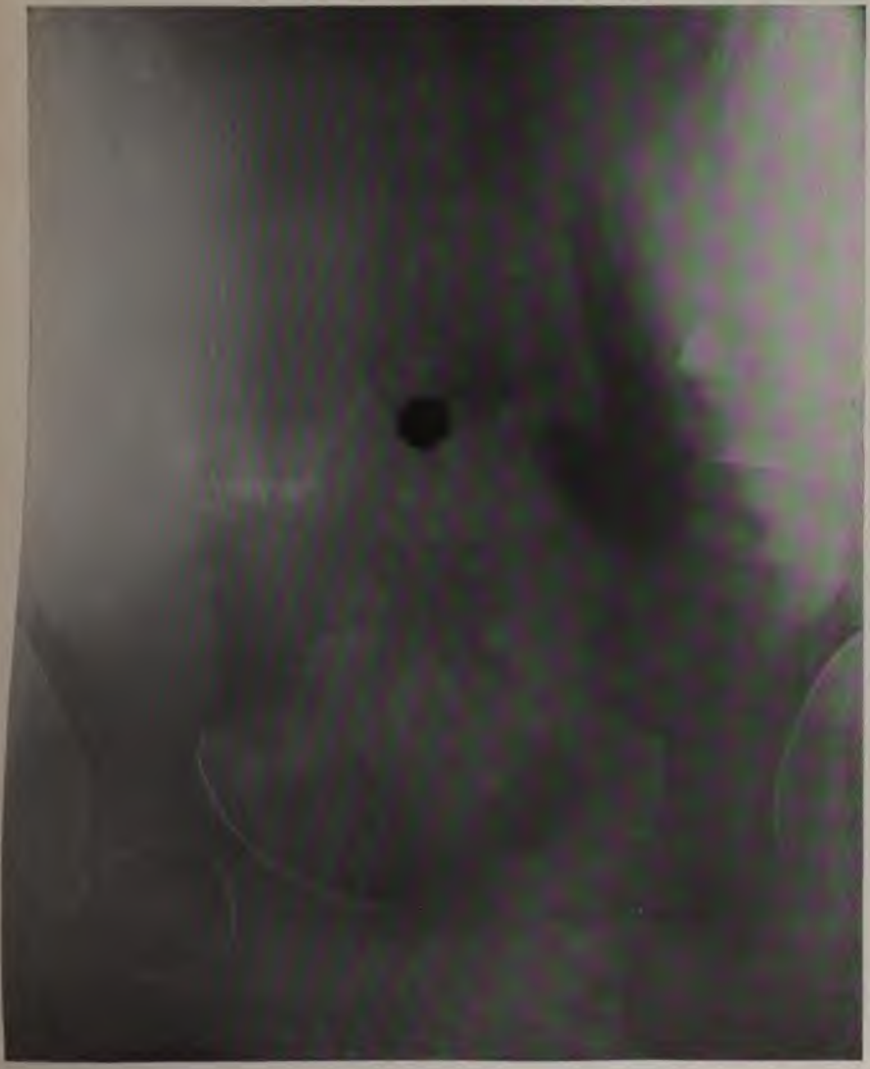
PLATE III



Same patient examined in the standing position, just after taking the food. Shadow of the cent on the umbilicus. The stomach has now fallen entirely below the umbilical line. Notice the empty descending colon just to the left of the stomach. The negative shows the ascending colon empty, too, but it is not reproduced in the print.

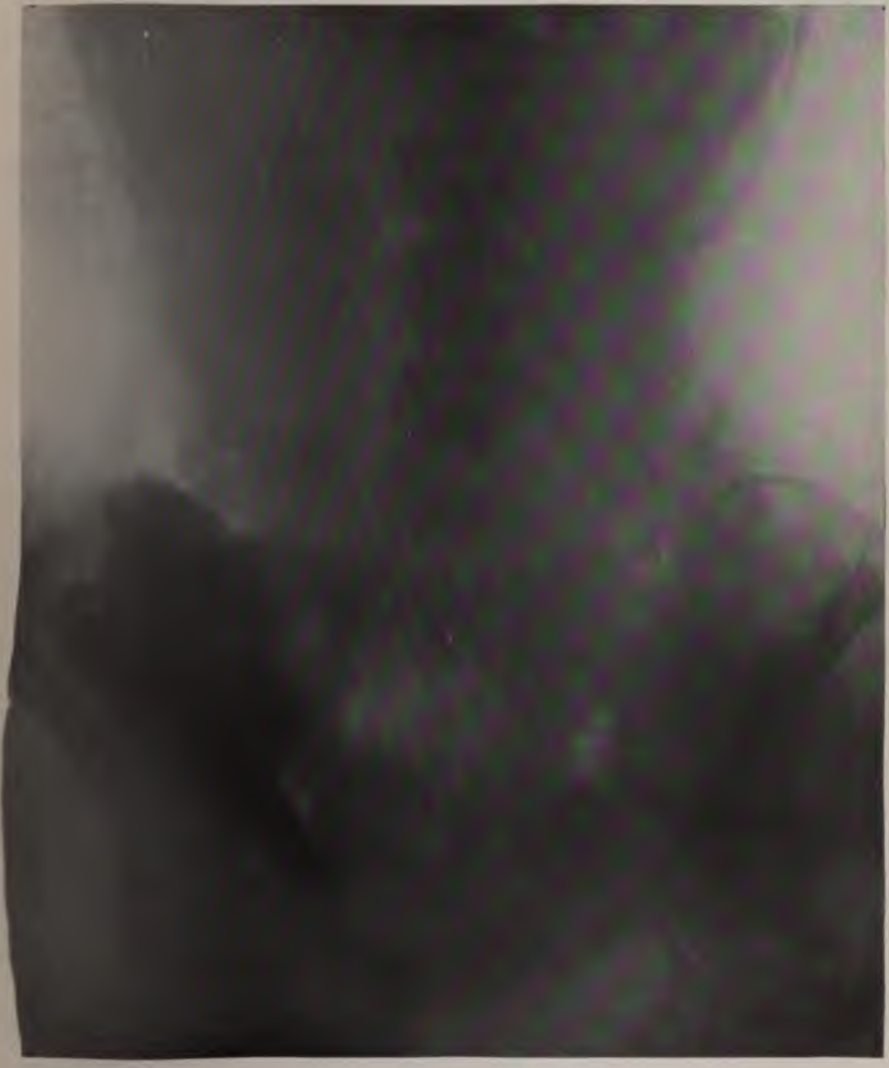
(Service of Dr. W. E. Ashton. Skiagram by Dr. G. E. Pfahler.)

PLATE IV



Same patient examined in the standing position six hours after taking the bismuth food. Half of the food is still remaining in the stomach, indicating delayed motility. The remainder of the food is scattered through the small intestines. (Service of Dr. W. E. Ashton. Skiagram by Dr. G. E. Pfahler.)

PLATE V



Same patient examined twenty-four hours after taking the bismuth food. Shows the food in the cæcum. Notice the constriction in the lower portion of the shadow which indicates the ileo-cæcal valve. A negative made in thirty-six to forty-eight hours will usually show the food in the transverse colon. In this case a negative made forty-eight hours after taking food showed only a small portion of the food remaining in the rectum, the balance having been passed by bowels. (Service of Dr. W. E. Ashton. Skiagram by Dr. G. E. Pfahler.)

Photography of the Stomach.

Repeated attempts have been made in recent times, at first by R. Kuttner,[127] next by Lange and Meltzing,[128] and more recently by Bial,[129] to photograph the interior of the stomach. Kelling[130] attempted also to photograph the œsophagus of animals and human beings, and succeeded in obtaining some interesting results. None of these attempts have as yet been followed by any practical results.

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[127] R. Kuttner, Deutsche medicinische Wochenschrift, 1891, No. 48.

[128] Lange and Meltzing, Münchener medicinische Wochenschrift, 1898, No. 50.

[129] Bial. Centralblatt für die medicinischen Wissenschaften, 1899, No. 7.

[130] Kelling, Archiv für Verdauungskrankheiten, 1898, Bd. 3, Heft 3.

CHAPTER V.

Methods of Chemical Examination.

Examination of Mixed Saliva.

In examining the saliva we must take into consideration its quantity, reaction, condition of its ferment, and the presence of abnormal constituents.

The *quantity* of the saliva, which varies even under normal conditions, may be abnormally large or very small. The saliva is markedly increased in amount (collection of water in the mouth) in not a small number of stomach-diseases, this symptom being obviously reflex in nature. It can, however, be of no practical diagnostic use, for it may be met with in a large variety of diseases. Some diseases, on the other hand, are accompanied by a more or less marked disappearance of the salivary secretion. This is the case most frequently in gastric cancer and in catarrhal diseases of the mucous membrane of the stomach.

The two anomalies of sub- or super-secretion of the saliva are both found in the varied forms of nervous dyspepsia and enteropathic conditions.

The *reaction* of the saliva, as has been previously stated, is alkaline; its degree of alkalinity, however, fluctuates within wide limits. No diagnostic significance can be attributed to this phenomenon.

I have repeatedly observed an acid saliva, especially in hysterical women; this finding was so variable in cases of organic gastric disease that it was impossible for me to designate any distinct type. L'Héritier found the saliva constantly acid in five cases of gastric cancer, while Frerichs made a similar observation in six cases of ulcerating gastric cancer. Acid saliva was also found by Sticker[1] in two cases of cancer of the stomach; C. Hübner, on the other hand, is of the opinion that this condition of the saliva is not the rule. In six cases which were thereupon examined by me, I also noted an alkaline reaction in some and an acid reaction in others. The previous positive results may probably be accounted for by lack of oral cleanliness, which is absolutely necessary. In Sticker's two cases the reaction was due to lactic acid.

The *ferment* of the saliva in diseased conditions has hitherto received but little attention. It is therefore impossible to answer with certainty the question whether cases accompanied by disappearance or diminution of the amount of ptyaline really exist, which, analogous to secretory insufficiency of other glands, might be expected.

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[1] G. Sticker, Die Bedeutung des Mundspeichels in physiologischen und pathologischen Zuständen, Berlin, 1889, S. 23.

In my experience on this subject, which to be sure is limited, I feel assured that ptyaline secretion may be *deficient* in diseases of the stomach. Klemperer[2] incidentally reported a case in which the quantity of ptyaline was directly diminished. Jawein[3] also found a diminution of the ferment in chronic nephritis, ascites, scorbutus, diabetes, and Addison's disease. I believe the same condition existed in two cases in which, in spite of the absolute absence of hydrochloric acid in the gastric contents, large quantities of amidulin (soluble starch, which turned blue upon the addition of iodine) could be demonstrated in the stomach after a long interval of time. Examination of the saliva was unfortunately not performed.

On the other hand, the ferment seems, on the whole, to be fairly resistant to external influences. I have repeatedly examined the activity of the amylolytic action of the saliva in stomatitis, periodontitis, alveolitis, etc., and found it to be preserved. Accurate tests for sugar may, however, show variations from normal. The great demand made upon the glandular activity in ptyalism probably leads, as a rule, to a diminution in the amount of the ptyaline; I myself have nevertheless noted a good saccharificating function in a case of ptyalism, although no quantitative estimation was made. It follows, therefore, from the above discussion that it is important to test the saliva for its ferment. It is especially desirable to examine the saliva when a large amount of dextrine or starch is preserved in a gastric juice free of hydrochloric acid. The previous thorough cleansing of the mouth is also absolutely necessary in this case.

Qualitative changes of the saliva were frequently reported and given much consideration by earlier physicians. Wright, and with him, Wunderlich and Oppolzer, still assumed the existence of a solid, sweet, bilious, purulent, stinking, calcium-containing saliva. It is clear, on the whole, that an increase in the salivary secretion must be followed by a diminution of its solid contents, and especially of its organic constituents. L'Héritier's examinations of the saliva in chlorosis, albuminuria, pneumonia, erysipelas, and febrile inflammations demonstrated considerable differences in comparison with the healthy in regard to the presence of organic substances and salts. To what extent these findings are applicable to other conditions, and especially to disturbances of the digestive organs, cannot yet be answered, because of insufficient investigation on the subject.

This may also be said of the presence of the previously-mentioned *sulphocyanates* in the saliva, which were especially noticed in the matutinal vomitus. On the other hand, Fenwick[4] found only a small amount of potassium sulphocyanide, or its complete absence, in gastric carcinoma.

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[2] Klemperer, Verhandlungen des Kongresses für innere Medicin, 1889, S. 276.

[3] Jawein, Wiener medicinische Wochenschrift, 1892, No. 15 u. 16.

[4] Fenwick, Med.-chir. transaction, 1882, Bd. 55, S. 116.

Rosenbach[5] directs attention to certain colour-reactions of the saliva which are supposed to be of diagnostic value in certain forms of functional disturbances. If an excess of nitric acid is added to the saliva, a canary-yellow colour is produced, whereas the addition of alkalies in excess calls forth a brownish-yellow or orange-coloured tint. By gradually adding boiling hydrochloric acid in excess, one observes a rose colouration which, under certain circumstances, not yet exactly understood, passes into reddish violet, and upon the addition of caustic soda, into dark violet. Further boiling with hydrochloric acid converts the coloured substance into a slightly yellow tint, or even into a saturated yellow; and the addition of an excess of caustic soda produces a dark yellow, but never the above-mentioned brownish or reddish yellow.

Jul. Rosenthal,[6] who paid considerable attention to these colour-reactions, found that the rose-coloured reaction cannot be obtained in normal persons without marked irritation (for example, after smoking, use of condiments, injection of pilocarpine) of the salivary glands. On the other hand, this author found the colour-reaction in greatest intensity in carcinoma of the stomach and in marked nephritis.

Examination of the Chemical Functions of the Stomach.

1. Without the Use of the Tube.

The Vomitus.

The vomitus is sometimes of vast diagnostic importance, for by means of it a previously doubtful diagnosis may become absolutely clear, although in some cases it is of little value for that purpose. It is, however, an important symptom in all cases, and furnishes an opportunity for making a close investigation. We should note in examining the vomitus, as in the case of stomach-contents, the appearance, quantity, odour, taste, chemical condition, microscopical findings, and the admixture of abnormal constituents which may be present. Inasmuch as these points are mostly referred to in detail in the Chapter on Examination of the Stomach-contents, it remains only to discuss here the symptomatic significance of the vomitus.

First, it is of importance to determine whether the vomitus does or does not contain remnants of food.

In the former case it is important to inquire into the last and previous meals, as well as to the interval of time which has elapsed since the last meal; for thus valuable information may be obtained as to the course of digestion. *If, for example, distinct remnants of food eaten the previous days are found in the stomach-contents,* it is highly probable that a mechanical obstruction to peristalsis exists (stenosis of the pylorus, hour-glass constriction, or myasthenia, etc.). Intelligent patients frequently state that the

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[5] O. Rosenbach, Centralblatt für klinische Medicin, 1891, No. 8.

[6] Jul. Rosenthal, Berliner klinische Wochenschrift, 1892, No. 15.

vomitus shows the presence of gas, from which, in conjunction with the other findings of the examination, the diagnosis of severe motor insufficiency can be made, even without exploration with the tube. The quality of the vomitus is also significant; and we should therefore determine whether the food is digested, partly digested, or not at all digested, whether any difference exists between albumen and carbohydrate digestion, and whether large quantities of fat or fatty acids are present. If the ingesta is well digested, the vomiting is probably due to a gastric neurosis, or it may be of reflex, cerebral, or spinal origin. The vomitus may, however, also consist of pure chyme in organic diseases of the stomach, as, for example, in the beginning stages of gastric ulcer and in catarrhal gastritis. Conclusions cannot, however be drawn as to the state of the digestion because of the presence of insufficiently digested food in the vomitus, for large quantities of mucus may have been either produced or swallowed previous to the vomiting act, and thus interfered with digestion. If the vomiting is not preceded by nausea, we may find in the quality of the vomitus a criterion for anomalies of digestion, which is not to be underrated. In that event the presence or absence of mucus and saliva will account for that phenomenon, even though we must be cautious in arriving at such a conclusion. If the vomitus shows no signs of digestion whatever (apepsia), particularly when a long period of time (two to three hours after a small and four to five hours after a large meal) intervenes between the last ingestion of food and the time of vomiting, it is highly probable that a marked disturbance of the glandular apparatus exists. In case of a mixed diet, however, it is not always so simple to determine whether actual apepsia exists. A few undigested remnants of meat may, for example, be found in a stomach whose chemical activity is satisfactory; only the presence of an abnormally large quantity of meat in such cases can be considered indicative of apepsia. The vomitus may be mixed with abnormal products, consisting chiefly of *blood,* rarely *pus, mucus, saliva, bile, intestinal juice,* or *intestinal contents, particles of tissue from mucous membrane or tumours, fæcal material, segments of parasites,* etc.

(*a*) *Blood.*—Blood contained in the vomitus does not always originate in the stomach. It is more likely to arise during the act of vomiting, from the œsophagus, trachea, pharynx, or oral cavity. One should therefore be cautious in designating the significance of such hæmorrhages. The blood, as a rule, undergoes a decided change, due to the influence of the vomited material, although occasionally it remains fresh. Conclusions may occasionally be drawn from the condition of the blood as to the character of the vomitus. If after standing several hours the vomitus still shows pure blood and the microscope demonstrates the presence of well-preserved erythrocytes, a pronounced production of acid in the stomach is out of the question; and *vice versa.* The altered hæmoglobin undergoes certain colour-changes, varying from dark black to the appearance of coffee-grounds or cocoa, depending upon the character of the vomitus and also upon the length of time it is mixed with the stomach-contents. The coffee-ground appearance, provided it is present immediately after vomiting, is always indicative of long-continued sojourn in the stomach. Slight hæmorrhages, as has already been noted by L. Kuttner,[7] and as I have been able to confirm in numerous cases, alter in no way the vomitus or the stomach-contents. Hence in such cases a thorough chemical and microscopical examination is necessary (see Chapter on Examination of Gastric Contents).

(*b*) *Pus.*—A more or less large amount of pus is mixed with the vomitus in rare cases (phlegmonous gastritis, ulcerative diphtheritic carcinoma, gastric abscess, etc.). One look through a microscope will establish the diagnosis, if the presence of pus is doubtful macroscopically. The source of the pus can, however, by no means be determined in this manner, for it may come from the air-passages, the pharynx, or the nasopharynx; likewise it may originate in an abscess of the large intestine, *which led to adhesions* with the stomach (gastrocolic fistula); then, again, it may arise in a suppurative pancreatitis, a liver-abscess, and, finally, in the small intestines. For further consideration of this subject consult Chapter on Examination of Gastric Contents.

(*c*) *Mucus* and *saliva* are always present in the vomitus. Large quantities are easily recognised, while abnormally small amounts can be demonstrated by means of the microscope (see below).

(*d*) *Bile* is likewise not rarely present in the vomitus. It can, however, only be considered of diagnostic significance when *constantly present in large amounts* (see below). In order to detect bile in doubtful cases, it is necessary to resort to the biliary pigment-test (Gmelin's test), as well as to the (after removal of the albumen by means of phosphor-tungstic acid) Pettenkofer test for biliary acids, or by demonstrating the presence of cholesterin.

(*e*) *Particles of mucous membranes or tumours* are found in the vomitus in rare cases, microscopical examination of which may, under certain circumstances, reveal important information. For further discussion of this topic see microscopy of the gastric contents.

(*f*) *Fæcal vomiting* may be detected by its odour. If a large amount of fæcal substances is present, a distilled specimen will show the presence of phenol, indol, and skatol.

(*g*) *Parasites.*—Those found at times in the vomitus include ascarides, segments of tænia, oxyuris vermicularis, anchylostomum duodenale, and trichinæ. Hydatid cysts of echinococcus have also been observed after rupture of the liver into the stomach. Those rarely met with are maggots (Meschede), larvæ of diptera (Gerhardt), and fly larvæ (Küchenmeister, Lublinski, Senator, and others).

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[7] L. Kuttner, Berliner klinische Wochenschrift, 1895, No. 7 and 8.

If the vomitus *contains no food,* we may find either blood, saliva, mucus, bile, or intestinal juice. The vomiting of blood occurs in ulcer, under certain circumstances also in cancer, cirrhosis of the liver, varicose degeneration of the œsophageal and gastric veins, congestion of the liver, particularly in icterus, or also in cardiac valvular diseases, with marked stasis in the lesser circulation. The most profuse hæmorrhages originate, as a rule, in the stomach or in the duodenum; yet the interesting statistics of Quincke[8] show that a large amount of blood may also arise in ulcer of the œsophagus (generally near the cardia). Ulcerating cancer of the œsophagus may also occasionally account for a considerable amount of blood in the vomitus. When the blood is due to other causes, the lungs, liver, and heart must also be examined. Hæmophilia, which was the cause in one of my cases, must be thought of. The diagnosis of *vicarious* gastric hæmorrhages can only be made when disturbances of menstruation actually exist, and then only with great caution. [Rodman,[9] in his excellent oration on Gastric Hæmorrhage, mentions post-operative hæmorrhage, hæmorrhage in purpura hæmorrhagica, bleeding from miliary aneurism of the aorta and other vessels, and in leukæmia, typhoid, yellow, and other infectious fevers, and various kinds of traumatism.]

Many physicians are inclined to accept slight gastric hæmorrhages (one to two teaspoonfuls of blood) as a sure sign of ulcer. I have had the opportunity of observing many cases in which, even though disturbances of digestion already existed, further examination revealed absolutely no manifestation of ulcer. We may realise how unjustified is the diagnosis of round ulcer under such circumstances, when we consider the ease with which small capillaries bleed when irritated by a tube, food, or forcible pressure, and that, too, without any ill effect upon the patient. A similar observation was made by L. Kuttner,[10] who based his results on abundant material. On the other hand, the constant presence of minute quantities of blood may confirm a doubtful diagnosis.

The vomiting of pure *saliva* is a frequent symptom in chronic catarrh of the pharynx, which, on account of the swallowed saliva, is not rarely followed by a secondary catarrh of the stomach. The vomitus in this case is a thin, liquid mass, which contains but a small amount of solid substance and whose specific gravity varies from 1004 to 1007. This form of vomiting is commonly designated as the vomitus matutinus, or *water-brash.* Its reaction is alkaline, as a rule. Characteristic of this vomitus, aside from the mucin reaction, is the Burgundy-red colour obtained upon the addition of a dilute solution of the chloride of iron, which is indicative of the presence of sulphocyanates.

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[8] Quincke, Deutsches Archiv für klinische Medicin, 1879, Bd. 24, S. 72.

[9] [Rodman, Philadelphia Medical Journal, June 9, 1900.]

[10] L. Kuttner, Berliner klinische Wochenschrift, 1895, No. 7-9.

The *vomiting of mucus,* provided it occurs repeatedly, signifies an existent chronic pharyngitis or gastric catarrh, or both. Careful inspection of the pharynx, as well as exploration of the stomach with a tube, leaves no doubt as to the diagnosis. The mucus is occasionally mixed with blood and bile, and, as a rule, with undigested food-remnants. Vomiting of pure mucus speaks, in my experience, almost surely *against gastric dilatation.* The quantity of mucus vomited is generally very slight—that is, from one to two tablespoonfuls, or even less.

Vomiting of bile or intestinal juice occurs either acute (for example, in acute gastritis, peritonitis, perityphlitis), or it is a temporary manifestation of various chronic gastro-enteric diseases, or it may occur as a reflex phenomenon in diseases of the liver, kidneys, ovaries, uterus, and in tabes (gastric crisis), etc. Of diagnostic importance is the *constant occurrence of bile in the vomitus or in the stomach-contents.* It is highly probable, then, that its presence is due to stenosis of the descending portion or the horizontal inferior portion of the duodenum or the commencement of the jejunum (Leichtenstern,[11] Riegel,[12] Cahn,[13] Honigmann,[14] Hochhaus,[15] Boas,[16] Reiche,[17] Schüle,[18] Herz,[19] Wegele,[20] and Rewidzoff[21]). Bile tends always to be mixed with pancreatic juice provided the pancreas is intact, so that when it is *constantly absent* in a bile-containing vomitus, disease of the pancreas or displacement of the duct of Wirsung may be suspected (Boas[22]).

Although under certain circumstances careful examination of the vomitus will reveal valuable information, yet the latter must be accepted *with great caution in estimating the chemical functions of the stomach.* We must not lose sight of the fact that vomitus, at least when due to gastric disease, is the manifestation of *abnormal chemical or mechanical irritation of the gastric mucosa.* The fact that vomiting occurs is sufficient evidence that temporary abnormal changes must have previously occurred in the composition of the stomach-contents.

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[11] Leichtenstern, Verengerungen, Verschliessungen und Lageveränderungen des Darmes, v. Ziemssen's Handbuch der speziellen Pathologie und Therapie, 2 Aufl., Bd. 7, 2, S. 411 u. 418.

[12] Riegel, Zeitschrift für klinische Medicin, 1886, Bd. 11, Heft 2 u. 3, S. 167; Deutsche medicinische Wochenschrift, 1890, No. 39.

[13] Cahn, Berliner klinische Wochenschrift, 1886, No. 22.

[14] Honigmann, Berliner klinische Wochenschrift, 1887, No. 18.

[15] Hochhaus, Berliner klinische Wochenschrift, 1891, No. 17.

[16] Boas, Deutsche medicinische Wochenschrift, 1891, No. 28.

[17] Reiche, Jahrbücher der Hamburger Krankenanstalten, S. 180 u. f.

[18] Schüle, Berliner klinische Wochenschrift, 1894, No. 45.

[19] Herz, Deutsche medicinische Wochenschrift, 1896, No. 23-24.

[20] Wegele, Münchener medicinische Wochenschrift, 1898, No. 16.

[21] Rewidzoff, Archiv für Verdauungskrankheiten, 1898, Bd. 4, S. 369.

[22] Boas, *l.c.*

Direct Examination of the Gastric Secretion.

On account of the inconveniences associated with the passage of a tube, numerous authors have described various methods by means of which the secretion of the mucous membrane can be examined in a simple manner. The same have, however, not been approved in practice, and I only present them here in order to be complete.

1. *Method of Edinger.*[23]—This author recommends the swallowing of small sponges compressed into a gelatine capsule, which are attached to silk threads. The capsule is rapidly dissolved, and after the sponge has become saturated with gastric contents it is withdrawn by means of the silk thread. This method has the disadvantage that as the sponge is pulled out it absorbs mucus, which neutralises the gastric juice.

2. *Method of Späth.*[24]—The latter employs silk threads fastened to little globules made of elder-marrow (pith), saturated with a solution of Congo-red, which turns blue if hydrochloric acid is being secreted. This method is only of value when reaction is positive; a negative result may also be caused by the subsequent effect of mucus, saliva, etc. (See Chapter on Detection of Hydrochloric Acid.)

3. *Method of Günzburg.*[25]—Günzburg makes use of potassium iodide wrapped in very thin rubber tubing, which has a tendency to burst. A tablet of 0.2 to 0.3 gramme of potassium iodide is pushed into such a tube, the two ends of which are closed by folding them backward until they meet each other. The ends are tied in place by means of three fibrine threads, which have been previously preserved in alcohol and thus hardened. A package prepared in this manner and previously placed in glycerine is forced into a gelatine capsule and swallowed one hour after a test-breakfast. The saliva of the patient is then examined every fifteen minutes with the starch-test for the presence of potassium iodide.

The theory of this method consists in the fact that the time required for the appearance of the iodine reaction depends upon the digestive energy, the latter being measured by the time required for the fibrine to dissolve or swell. The objections to this ingenious method are due to the impossibility of determining the exact site of the dissolution (stomach or intestines), and to the fact of the presence of fermentation acids, which cause a total or partial swelling of the fibrine threads, and thus lead to a simulation of the normal iodine reaction. Günzburg's method was tested by Marfan,[26] who essentially confirmed the researches of that author. Bäklin, working in my laboratory, on the contrary, found that the potassium iodide reaction may be delayed in cases of excellent gastric secretion, thus causing the expectation of hydrochloric acid insufficiency, when hydrochloric acid is, as a matter of fact, present in sufficient amounts. On the other hand, Bäklin was not able to substantiate the objection raised by Sahli[27] against Günzburg's method, namely, that the fibrine threads swell even in water so that the presence of potassium iodide may be demonstrated a few minutes after the packet has been placed in that liquid.

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[23] Edinger, Deutsches Archiv für klinische Medicin, 1881, Bd. 29.

[24] Späth, Münchener medicinische Wochenschrift, 1887, No. 41.

[25] Günzburg, Deutsche medicinische Wochenschrift, 1889, No. 41.

[26] Marfan, Archives générales de medecine, May, 1890.

Sahli elaborated a similar method even before Günzburg, whose object, however, was not to test the hydrochloric acid secretion, but to seek information on the whole course of digestion. This method is as follows: A pill containing 0.2 gramme potassium iodide, prepared in the usual way, but with the addition of a little glycerine (to prevent drying), is wrapped in a very thin (as thin as tissue paper) piece of Para rubber about the size of a silver dollar; it is so wrapped that the free edges of the small piece of rubber can be twisted tightly together over the pill in a pouch-like manner. It is then placed in a gelatine capsule and swallowed. The time at which iodide appears in the saliva is noted. Henne[28] employed this method in a series of interesting investigations on the action of certain therapeutic agents.

4. *Method of Einhorn.*[29]—Two silver spherical capsules are so fitted together that the larger, which is the receptacle, surrounds the smaller, which represents the cover. The latter is fastened to a thread, which is swallowed into the stomach with this small apparatus (1¾ centimetres long, ¾ centimetre wide) attached; as soon as the lower capsule finds ground on which to rest, the cover falls off by its own weight and permits the stomach-contents to enter the receptacle; upon withdrawal, the cover closes the opening of the spherical capsule and prevents the admixture of mucus to the gastric contents. The apparatus has, however, not proven practicable, and Einhorn has therefore constructed a small silver bucket which is employed to scoop the stomach contents, in order to examine them for free hydrochloric acid and the rennin-ferment.

5. *Method of F. Reach.*[30]—A mixture of an iodide with an iodate (barium iodide and bismuth oxyiodate) is resorted to by this investigator to detect free hydrochloric acid. The patient swallows, at the end of a test-meal, a gelatine capsule containing 0.6 gramme of a mixture of barium iodide and bismuth oxyiodate in the proportion of 1 to 2. The patient is then requested to spit from time to time into a vessel. It remains then only to test for iodide. If free hydrochloric acid is absent, the iodine reaction is much delayed in appearance; whereas in superacidity it occurs very rapidly (in thirty minutes). The iodine reaction may, however, be delayed in marked motor-disturbance, even though the secretion of hydrochloric acid is normal or increased. It follows, therefore, that this method is of no practical value for testing the secretion.

2. With the Use of a Tube.

Methods for Obtaining the Stomach-Contents.—The stomach-contents can be obtained in two different ways; that is, it can be aspirated, or it can be expressed by exerting pressure upon the abdomen. It is necessary, for practical purposes, to become acquainted with both methods, for occasionally one may be supplemented by the other.

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[27] Sahli, Korrespondenzblatt für Schweizer Aerzte, 1891.

[28] Henne, Zeitschrift für klinische Medicin, 1891, Bd. 19, Suppl., S. 286.

[29] Max Einhorn, New York Medical Record, July, 1890.

[30] F. Reach, Fortschritte der Medicin, 1898, Bd. 16, No. 19.

(*a*) *Aspiration Method.*—This procedure may be practised by means of the *stomach-pump,* as it was first proposed by Kussmaul and frequently modified later. It is subjected to many disadvantages from a practical point of view. These include the fear patients feel toward the application of an instrument (the term "pumping out the stomach," which unfortunately is still employed in medical circles, is frequently sufficient to frighten a patient*); the instrument usually employed is also quite complicated and is taken apart with difficulty, while its component portions are hard to clean. By no means least in importance is the danger of tearing off large sections of mucous membrane, which is known to have occurred on application of the stomach-pump as well as in simple expression (v. Leube,[31] Malbranc, v. Ziemssen,[32] Crämer,[33] Ebstein[34]). According to my abundant personal observations, this accident has never had any untoward effect upon the patients, the sections of injured mucosa being, however, in these cases comparatively small. I cannot understand, therefore, how Fleiner[35] could designate this most harmless occurrence as "malpractice," in spite of the fact that, in my experience, this injury cannot always be avoided, even though caution be exercised.

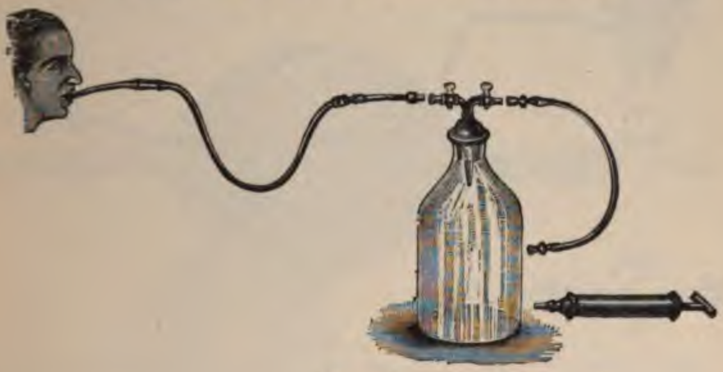

Fig. 22.

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* *I wish to urgently advise, on the grounds of extensive practical experience, against the employment of the expression "pumping out the stomach," which has now become obsolete. The extraordinary imaginations associated with this undertaking have cost many patients one or more sleepless nights.* Numerous patients would be willing to submit to an examination of the stomach-contents, but at no price would they consent to the "pumping out of the stomach."

[31] v. Leube, Deutsches Archiv für klinische Medicin, 1876, Bd. 18, S. 496.

[32] v. Ziemssen, Deutsches Archiv für klinische Medicin, 1872, Bd. 10, S. 66.

[33] Crämer, Münchener medicinische Wochenschrift, 1891, No. 52.

[34] Ebstein, Berliner klinische Wochenschrift, 1895, No. 4.

[35] Fleiner, Lehrbuch der Krankheiten der Verdauungsorgane, Stuttgart, 1896.

Aspiration may also be accomplished by means of *aspirator-flasks* for which the Potain apparatus employed for aspiration of pleuritic exudates serves as a model. Such apparatus has been largely modified to suit the purposes of diagnostic examination of the stomach-contents. A useful aspirator can be constructed in various ways.

Thus, analogous to the apparatus recommended by Fürbringer[36] for removal of the exudate in the pleural cavity, one may employ a Wulffian flask, or a glass flask whose stopper contains two perforations; two glass tubes as wide as possible are introduced into these openings, one of which connects by means of a rubber tube with the stomach-tube, while the other is joined to a Potain syringe or a rubber balloon, which exhausts the air. (Figs. 22 and 23.)

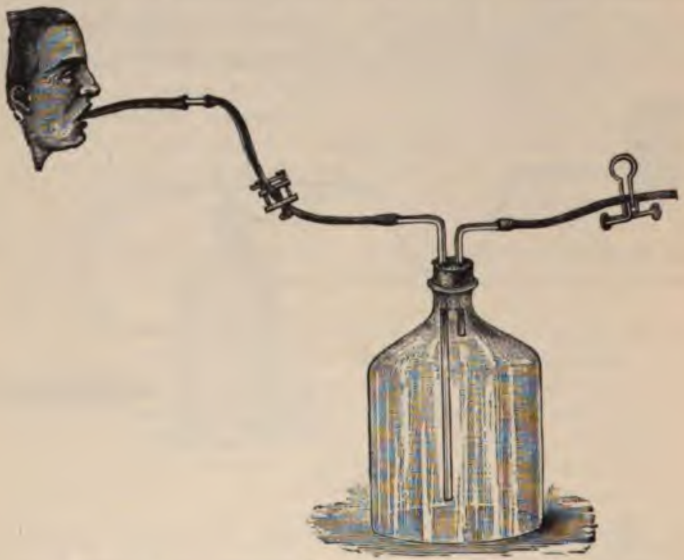

Fig. 23.

Aspiration by Means of an Aspirator-Bulb.—For this purpose a bulb in the form of a pear may be employed, as is done by Ewald,[37] which, like the one supplied to a Politzer apparatus, is attached to the stomach-tube in a suitable manner. In this case, however, the bulb must be emptied after each suction, which is very inconvenient.

I have used for years an aspirator (Fig. 24), which consists of a strong rubber bulb, which terminates on both sides in equally long rubber tubes.

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[36] Fürbringer, Berliner klinische Wochenschrift, 1888, No. 13, S. 254.

[37] Ewald, Klinik der Verdauungskrankheiten, 1893, 3 Auflage, S. 13.

The apparatus is connected with the stomach-tube by means of a connecting glass tube. The tube which does not connect the stomach-tube with the aspirator is also supplied with a clamp (Q), which closes its lumen. When the clamp is opened and the bulb is compressed, it is relieved of its air; if now the clamp is closed, the stomach-contents will be sucked up by the bulb. Upon opening the clamp, the contents of the bulb can be expelled, by compression, through the tube not connected with the stomach-tube, into a vessel which is held beneath it, after which the process is begun anew in a similar manner. The apparatus, inasmuch as it consists of one piece, can be thoroughly cleaned. After many years of use I can strongly recommend it.

[The apparatus which I use for aspiration of stomach-contents is a very simple one and can be kept absolutely clean, as the stomach-contents come in contact only with the stomach-tube and with glass. The exhaust-bulb itself is never in contact with the contents, and only air passes through the bulb. A bottle with a wide neck, in which the rubber stopper fits, can readily be obtained from any druggist. No stop-cocks are necessary, as the aspiration (and inflation) are regulated by the valve of the bulb. By reversing the bulb, so that the other small rubber tubing is connected with the glass tube in the rubber stopper, we may inflate the stomach, and so we get one and the same apparatus for aspiration and inflation. The bottle may be readily graduated by the physician himself by means of a file. Such a graduated bottle directly shows the amount of stomach-contents removed.]

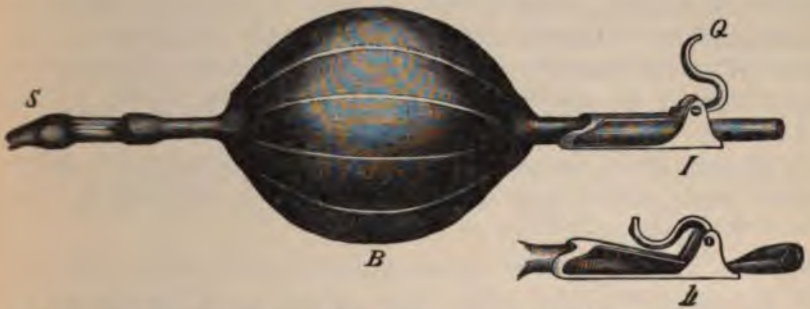

Fig. 24.—The Aspirator of Boas, for Removal of the Stomach-contents. *B*, Balloon. *S*, Tube. *Q*, Clamp, which is opened at *I* and closed at *II*.

More complicated are the apparatus originally proposed by Jaworski and Cyrianski. I will not discuss them, because they can be credited with no practical value and possess the disadvantages of all the less portable instruments.

(*b*) *Methods of Expression According to Ewald and Boas.*[38]—This is the simplest and most natural method of obtaining the stomach-contents. No other instrument than a stomach-tube, and no other requirement on the part of the patient than the exercise of moderate abdominal pressure, which may be assisted by coughing, are necessary. It is particularly useful to practitioners who do not always have the above-described instruments at their disposal. The expression is either performed easily or with great difficulty, and occasionally, indeed, it cannot be done at all, depending upon whether the stomach is filled with a large quantity of ingesta, and particularly of liquid contents, or with a thick chyme containing undigested remnants of food. The inability to obtain the contents by expression may be due to the plugging of the tube by means of thick undigested particles of food. If this be suspected, the passage may be easily re-established with a double bulb. If negative results are obtained by this method, it may be assumed, as a rule, that the stomach is empty. In order to make sure, this conclusion may be corroborated by performing a test-lavage. Since its introduction, the expression method has become the normal procedure for examination of the gastric contents, and has almost entirely supplanted the aspirator method.

[I have found that the majority of patients prefer the aspiration method, as I practice it, to the expression method, especially as it means less work for the patient.]

Examination of the Contents Obtained from the Fasting Stomach.

Examination of the fasting stomach is among the most important of the auxiliary methods for diagnosis. It alone is sometimes sufficient to make the diagnosis (for example, ectasia due to stenosis). Since the fasting stomach is empty, as a rule, the *finding of large quantities of contents* (over 50 to 100 cubic centimetres), *whatever its character may be, is to be regarded as anomalous.* However, as has been found by Schreiber[39] and confirmed by Rosin,[40] Hoffmann,[41] Pick,[42] Martius,[43] and Schüle,[44] good digestive secreted juice may be found in the fasting stomach in such quantities that pathological limits are almost reached. Such cases are exceptional when, according to my experiences, which coincide with those of Riegel,[45] Gintl,[46] and v. Aldor,[47] we consider that 5 to 20 cubic centimetres may be expelled from the secretory glands by the pressure itself; the irritation caused by the tube in such cases needs also to be considered. v. Aldor states also that the fasting secretion found in such cases is not true digestive gastric juice, for it possesses no pepsine. In regard to the minimal value of gastric secretion, which is to be considered in this connection, Schreiber, in my opinion, goes to extremes in claiming that a normal gastric juice is constantly present in the stomach.

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[38] Ewald und Boas, Virchow's Archiv, 1885, Bd. 101, S. 330.

[39] Schreiber, Archiv für experimentelle Pathologie, 1888, Bd. 24, S. 365; Deutsche medicinische Wochenschrift, 1893, No. 29 und 30.

[40] Rosin, Deutsche medicinische Wochenschrift, 1888, No. 47.

[41] A. Hoffmann, Berliner klinische Wochenschrift, 1888, No. 12 und 13.

[42] A. Pick, Prager medicinische Wochenschrift, 1889.

[43] Martius, Deutsche medicinische Wochenschrift, 1894, No. 32.

[44] Schüle, Berliner klinische Wochenschrift, 1895, No. 51.

The fasting stomach may contain:—

(*a*) *Remnants of food* from the last meal, or even from the one preceding it. If these are present in large quantities, and especially if much sour liquid, having the odour of fatty acids, is found in the stomach, it is a sure sign of a high-grade disturbance in the motility of the stomach. A few remnants which, lying in the folds of the mucosa, escaped expulsion into the stomach (especially frequent are fragments of hard-boiled eggs, fruits, raisins, plums, orange-peel, remnants of asparagus, etc.) are not, without further investigation, to be regarded as significant of a pathological condition.

Careful examination should reveal the character of the stomach-contents (kind of acids, micro-organisms, gastric fermentation, blood, mucus, etc.).

(*b*) *Pure Gastric Juice.*—Under certain circumstances, as was stated above, small quantities of gastric juice, which subjectively are not perceptible, may be expressed from either the healthy or the diseased stomach. If this quantity measures constantly 100 cubic centimetres or more, we may assume that a secretory anomaly exists; this condition is referred to as *gastrosuccorrhœa* (Reichmann) [gastrochylorrhœa]. Chronic gastrochylorrhœa is generally associated with a disturbance of the motor-function of the stomach, and indeed recent investigations (Schreiber,[48] Boas,[49] Rosenheim,[50] Hayem[51]), have shown that it is highly probable that gastrochylorrhœa is practically a sequel of the latter. Even when gastrochylorrhœa is found in gastric ulcer, the constant presence of a more or less pronounced disturbance of motility is unmistakable. As a rule, gastrochylorrhœa is combined with the secretion of an excessive amount of acid gastric juice (superacidity). The gastric juice is examined chemically and microscopically, according to the rules given in the Chapter on Examination of the Gastric Contents.

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[45] Riegel, Die Erkrankungen des Magens, Wien, 1897, S. 382 u. f.

[46] Gintl, Münchener medicinische Wochenschrift, 1897, No. 23.

[47] v. Aldor, Berliner klinische Wochenschrift, 1901, No. 39.

[48] Schreiber, Deutsche medicinische Wochenschrift, 1894, No. 18, 20, 21; Archiv für Verdauungskrankheiten, 1896, Bd. 2, S. 423.

[49] Boas, Berliner klinische Wochenschrift, 1895, No. 46.

[50] Rosenheim, Berliner klinische Wochenschrift, 1894, No. 50.

[51] Hayem and Lion, Traité de Médecine par Brouardel & Gilbert, 1897, Tome IV.

(*c*) *Mucus and Saliva.*—Aside from the admixture of small amounts of mucus and saliva to the fasting stomach secretion, which occurs physiologically, the stomach may also contain nothing but mucus or saliva. Stomach-contents of this type are characterised, aside from their well-known appearance, principally by their viscidity upon pouring from one vessel into another. Microscopical examination shows the presence of numerous mucin bodies and epithelium. The presence of mucus or saliva may be due to inflammatory processes in the mouth (stomatitis, ptyalism, pharyngitis, etc.), or to pathologic secretion of the mucous glands of the stomach (mucous catarrh), or finally to processes originating in these two conditions (for further information refer to the Chapter on Examination of the Gastric Contents).

(*d*) *Bile.*—Bile has been known to regurgitate into the stomach in many cases. After long-continued sojourn in the stomach, its bilirubin is converted into biliverdin, and the bile becomes yellowish or grass-green. Admixture of the gastric juice with bile or intestinal juice (see below, *e*) causes precipitation of biliary mucin and the *animal gum* (Landwehr), as well as the bile-pigments; these may again be dissolved by treating with alkalies. Bile does not interfere, as is still occasionally claimed, according to the views of Brücke, with the peptic activity of the gastric secretion, but, *like every other albuminoid body, it simply withdraws the acid.* If enough acid is secreted by the gastric mucous membrane to insure the presence of hydrochloric acid in the free state in the mixture of bile and gastric juice, the mixture will digest as well as pure gastric juice which contains a like amount of hydrochloric acid. The rennin-ferment of a mixture of bile and gastric juice is also preserved as long as free hydrochloric acid is present. *Accordingly, bile has no disturbing effect upon the gastric secretion.*

(*e*) *Intestinal Juice.*—Aside from bile we occasionally find in the fasting stomach, according to my observation,[52] a mixture of bile, pancreatic juice, and probably also succus entericus, which, for the sake of simplicity, I have designated as "enteric juice."

In the pure state, unmixed with gastric juice, it is grass-green in colour, absolutely clear in dilute solution, viscid and easily gelatinised, and possesses, aside from the constituents of bile, the biological properties of the pancreatic juice; that is, converts starch, raw as well as cooked, and likewise glycogen, first into maltose and then into dextrose, proteids into peptone (tryptone), gelatine into gelatine tryptone, and splits fats into glycerine and their respective fatty acids. In the presence of a large amount of Na_2CO_3 this mixture forms emulsions with oils or fats containing fatty acids, even without gross mechanical assistance; this is, however, no specific function of the pancreatic juice. Leucin and tyrosin are also easily demonstrable in all intestinal juices. The digestive property of the pancreatic juice is destroyed under the influence of great quantities of gastric juice; under such circumstances, as was stated above under the discussion of bile, the biliary mucin and the bile-pigment are precipitated and the biliary acids are set free. Aside from the mucin, the ferments of the pancreatic juice are also precipitated, and, if the action of the acids is prolonged (two to three hours), they are completely destroyed.

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[52] Boas, Zeitschrift für klinische Medicin, 1890, Bd. 17, Heft 1 and 2; Deutsche medicinische Wochenschrift, 1891, No. 28.

When the degree of acidity is slight, that is, when the mixture gives an acid reaction but contains no free acid, no precipitation whatever occurs; the mixture remains clear and the ferments are present in the active state.

We have already referred in detail to the diagnostic significance of the *constant* presence in the stomach of large amounts of intestinal juice mixed with bile. Small amounts of intestinal juice, whether obtained by expression or aspiration, are of no symptomatic significance.

(*f*) *Blood.*—Blood is found in the stomach under the following conditions: 1, irritation of an ulcer; 2, rupture of congested veins, as, for example, in cirrhosis of the liver and cessation of menses; 3, irritation of a pathologic or vulnerable gastric mucous membrane by the passage of a tube; 4, caused by vigourous movements of expression. It must not be forgotten that the hæmorrhages, as has already been stated, may also originate in the œsophagus, the nasal cavity, the pharynx, the lungs, etc. Hæmatemesis due to an ulcer can, as a rule, be easily distinguished from gastric hæmorrhage following other causes. In favour of the former are the characteristic anamnesis, the preceding disturbances, the epigastric and dorsal algetic points, the gastralgia, etc.; whereas in case of the latter, the factors causing the passive hæmorrhage (anomalies of the liver, heart, kidneys, and menstruation) are usually demonstrable. It is rarely difficult to differentiate between pulmonary and gastric hæmorrhages, for, aside from the characteristic condition of the blood, hæmoptysis (light red, foamy, tar-coloured stools never seen) will be revealed by the careful examination of the lungs. It must not, however, be forgotten that hæmoptysis and hæmatemesis may exist simultaneously. Slight hæmorrhages, the blood of which is generally mixed with mucus, are only of significance when found on repeated examinations. It is wisest, however, in such cases, as has already been stated, to desist in the use of a tube.

For the detection of blood in the stomach-contents, see the Chapter on Examination of the Gastric Contents.

(*g*) *Pus.*—Recent researches show that pus is frequently met with in the stomach-contents. I have found it most frequently in ulcerating carcinoma, in which condition it may be recognised even macroscopically by the foul-smelling, yellowish-green, and occasionally blood-stained masses. The diagnosis of ulcerating carcinoma, even though no tumour is found, may be based upon the presence of the latter, in connection, of course, with the other factors.

(*h*) *Fragments of Mucous Membrane.* (*i*) *Particles of Tumour Tissue.* (*k*) *Protozoa.*	Refer to microscopical Examination of the Gastric Contents.

Testing the Gastric Secretion After the Action of Various Stimulants.

A. Thermal Irritation.

According to v. Leube,[53] we may employ ice water for this purpose. One hundred cubic centimetres of ice water are introduced into the fasting stomach of an individual, and ten minutes later three hundred cubic centimetres of water are introduced and the contents removed and tested as to its reaction. This is performed as follows: 40 cubic centimetres of the acid stomach-contents, together with a small piece of hard-boiled egg, are placed in an incubator at a temperature of 35° to 40° C., and the time noted when the albumen is dissolved; if the reaction of the stomach-contents is neutral, it must be acidulated with hydrochloric acid.

Similar to this method is that of Jaworski.[54] Two hundred cubic centimetres of distilled water cooled with ice are poured into the fasting and empty stomach through a tube; at the end of ten minutes, the stomach-contents are aspirated without introducing a diluting fluid, and the filtrate is tested chemically and the flocculent precipitate microscopically. Neither method has obtained any foothold in practice. The irritation of ice water can furthermore not be employed in all diseases.

B. Chemical Stimulation.

Jaworski[55] was the first to point out that the introduction of dilute hydrochloric acid into the stomach was an excellent method for determining the presence of ferments in the stomach. This procedure may also be employed as a useful auxiliary measure for the study of the glandular secretion, and indeed it offers certain advantages over the digestive method. Its execution, which is accomplished far easier than the other method, is performed as follows: 100 cubic centimetres of a decinormal solution of hydrochloric acid is poured into the fasting stomach through a tube. The stomach-contents are aspirated or expressed ten minutes later. The liquid thus removed, which is generally somewhat turbid on account of flakes of mucus, is carefully filtered and then immediately titrated in the usual manner. Another portion is subjected to tests for digestion, examined for rennin ferment, etc., and the deposit on the filter is examined microscopically. (For greater detail, refer to Chapter on Examination of the Gastric Contents.) This procedure has not been adopted in the diagnosis of stomach diseases.

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[53] v. Leube, Deutsches Archiv für klinische Medicin, 1883, Bd. 33, S. 3.

[54] Jaworski, Zeitschrift für klinische Medicin, 1886, Bd. 11, S. 50.

[55] Jaworski, Deutsche medicinische Wochenschrift, 1887, No. 36-38.

C. Electrical Stimulation.

Ludwig and Weber, H. v. Ziemssen[56] and Bocci[57] were the first to show, by means of experiments on dogs, that the conduction of a strong faradic or constant current through the gastric wall is followed by secretion of gastric juice. The same phenomenon was observed by Regnard and Loye[58] in a person executed, by stimulating the vagi nerves with electricity forty-five minutes after death. A. Hoffmann[59] is credited with similar extensive and careful experiments on human beings. They were able to obtain through the stomach tube a by no means inconsiderable amount of gastric juice constantly after galvanisation of the stomach region; whereas the irritation of the tube produced either none, or only a very slight amount of weakly acid secretion. E. Goldschmidt,[60] on the other hand, contrary to these positive results, recently came to the conclusion that direct faradisation, as well as galvanisation, has no influence upon gastric secretion, even though strong currents are employed. The procedure is of no diagnostic value.

D. Digestive Stimulation.

Of the various stimulants to be considered and which will receive further discussion below, the digestive impulse is the most natural and *fulfils* most satisfactorily the actual conditions of the stomach. It offers an opportunity for obtaining a true and natural copy of the effect of the secretory, motor, and resorptive activity of the stomach, and by means of it any sensory disturbances, if present, will be ascertained.

The methods and details are more or less secondary in importance in comparison with this principle significant of the digestive stimulation.

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[56] v. Ziemssen, Ueber die physikalische Behandlung chronischer Magen- und Darmkrankheiten, Klinische Vorträge, Leipzig, 1888.

[57] Bocci, Lo Sperimentale, June, 1881.

[58] Regnard and Loye, Progrès médical, 1885, No. 28.

[59] A. Hoffmann, Berliner klinische Wochenschrift, 1889, No. 12 u. 13.

[60] E. Goldschmidt, Deutsches Archiv für klinische Medicin, 1895, Bd. 56, Heft 3 u. 4.

Every substance which nourishes a human being represents a true method for testing the chemical sufficiency, provided its action on the healthy stomach is known.

The further discussion entails the consideration of some extraneous factors, which, however, are of importance to the practitioner. These include: a uniformity, qualitatively and quantitatively, in the constituency of the test-meal; as far as is possible, prevent the patient from the arbitrary ingestion of any additional food whatever, and above all things select the plainest and simplest conditions; finally, the opportunity of obtaining sufficient amounts of gastric contents to enable one to study the course of digestion. Accordingly, no difficulty should be experienced in deciding which of the following methods of examination is to be employed. Following are the methods most frequently employed for this purpose:—

1. The Test-Breakfast of Ewald and Boas.

The patient eats in the morning, after having fasted all night, a roll of wheat bread (35 grammes) and drinks two cups of tea or, if an accurate chemical examination is to be made, 400 grammes of water. The stomach-contents are aspirated or expressed one hour later, and then examined for the presence of hydrochloric acid, pepsine, rennin-ferment, etc.

2. The Test-Meal of Riegel (Test-Dinner).

At noon the patient eats a meal consisting of a plate of beef-broth, 150 to 200 grammes of beefsteak, 50 grammes of purée of potatoes, and a roll of wheat bread. The stomach-contents are removed in three to four hours and examined for the points in question. This method has the disadvantage that particles of meat obstruct the lumen of the stomach tube* and increase the difficulty of removing the stomach-contents. Its greatest advantage consists in the opportunity it offers to note on first examination the degree of digestibility for starches and proteids. Similar to the Riegel test-meal is that proposed by Fleiner.

The acidity of the stomach after a test-meal and a test-breakfast has been studied by many authors (Jürgensen,[61] Riegel[62]). Jürgensen, as was perfectly natural, found increased acidity after a test-dinner. This does not make the latter preferable, for thus a pseudo-superacidity may be easily diagnosed. Of greater importance is the question whether free hydrochloric acid is found in either test. This is usually the case, according to Riegel. Only exceptionally is an abundance of free hydrochloric acid found by the test-breakfast, whereas it is absent, or present only in traces, after the test-dinner. On the other hand, Riegel has observed a case in which hydrochloric acid appeared only after the greater digestive stimulation of a test-dinner, while it failed to occur after the non-stimulating test-breakfast.

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* [This disadvantage can be readily avoided by ordering the meat in form of a broiled *scraped* beefsteak, whereby all tendons and other tough tissues are removed, and the patient must be told to masticate the food thoroughly.]

[61] Jürgensen, Berliner klinische Wochenschrift, 1889, No. 20.

[62] Riegel, Die Erkrankungen des Magens, 1897, und Münchener medicinische Wochenschrift, 1899, No. 45.

The following forms of test-meals are referred to only in order to be complete. None of them, in comparison with the above-mentioned tests, have received extensive application in the diagnosis of stomach diseases.

(a) Method of Klemperer.

The patient drinks half a litre of milk and eats two small rolls of wheat bread (milk test-breakfast). The stomach-contents are removed two hours later.

(b) Method of Germain Sée.

This author gives a meal of carefully scraped meat (60 to 80 grammes) and wheat bread (100 to 150 grammes). He requests the patient to masticate his food very thoroughly. The stomach-contents are tested two hours after ingestion.

(c) Method of Jaworski.

The stomach is first emptied, after which it is filled with 100 to 300 cubic centimetres of water which is again aspirated and the contents are examined in the usual way. After this preliminary test, the patient eats the whites of one or two boiled eggs and drinks 100 cubic centimetres of water. The stomach-contents are aspirated in certain intervals of fifteen minutes and examined for hydrochloric acid, digestive activity, etc. Normally, no albumen should be present in one hour and a quarter after its ingestion, and the liquid in the stomach should be clear or slightly opalescent, its reaction weakly acid or neutral, and no free hydrochloric acid should be present; this liquid should furthermore digest a small disk of albumen weighing 0.06 gramme in seven hours at the latest.

Aside from these methods, other test-meals or modifications thereof (for example, Bourget) have been recommended by certain authors (for example, Talma, Bouveret, etc.). We may refrain from describing them, since they offer in no respect any advantages over those mentioned above. It can not be denied, however, on the other hand, that our object may also be attained by means of these. Many incongruities in the secretion of hydrochloric acid in the stomach are also attributable to the various methods of different authors. The selection of a normal test-meal, as was very properly emphasised by Riegel,[63] is therefore very desirable in the interest of science.

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[63] Riegel, Die Erkrankungen des Magens, Theil 1, S. 87.

CHAPTER VI.

Examination of the Gastric Contents.

Macroscopic Examination of the Stomach-Contents.

Information of diagnostic value may be obtained even by the mere inspection of the stomach-contents. The *appearance, quantity,* and *odour* are to be taken into consideration.

Important conclusions may be drawn from the *appearance,* inasmuch as information is thus obtained as to the activity of the digestive juices. Thus we may distinguish between absolutely undigested, partially-digested, and well-digested contents.

The difference between carbohydrate and proteid (especially meat) digestion is also to be observed in this manner. Absolutely undigested stomach-contents, provided they are removed at the time recommended, occurs in cases of advanced catarrh, especially in atrophic conditions of the gastric mucous membrane and in the so-called achylia; in any case, it is indicative of serious secretory disturbance. The appearance of the test-meal, when removed in those cases, is exactly like that of such a meal placed in water; its filtrate is clear as water, which is a sign of the absence of peptic activity.

The admixture of abnormal substances is also to be noted. These include *blood, mucus, bile* or *duodenal juice,* and more rarely also pus, animal parasites, fragments of tissue from the mucosa and tumours, etc. Of importance is also the relationship between the solid substances and liquid. An abnormally large amount of liquid in comparison with a small quantity of chyme points toward pronounced retention of the former or to the secretion of fluids into the stomach. In marked gastric retention, the stomach-contents are not rarely observed to form into *three strata* when placed in a vessel. The upper layer is composed of mucus or undigested (or at any rate coarse) particles of food; the second layer, which, as a rule, is the largest, consists of fluid; while the lowest layer contains chyme. Such a formation of the stomach-contents denotes abnormal fermentation in the stomach, such as is found in the highest grades of mechanical insufficiency. On the other hand, when such a division of the stomach-contents into three strata does not occur, we can by no means exclude a disturbance of the motor apparatus. With reference to the quantity of the stomach-contents, an approximate idea as to whether the amount is normal or diminished (which suffices for general practice) can be obtained after repeated examinations following a test-breakfast. In my experience the filtrate of the stomach-contents which is obtained exactly one hour after a test-breakfast measures normally 20 to 50 cubic centimetres. This quantity may vary in two directions—that is, the quantity of the stomach-contents may be essentially diminished, or the stomach may be entirely empty. This condition of affairs, which is designated supermotility (hyperkinesis) of the stomach occurs in various nervous and organic gastric affections, especially in chronic gastritis, in achylia gastrica, in bulimia, and in diseases of the stomach which are accompanied by incontinence of the pylorus. In order to avoid error in such cases the stomach must by no means be emptied by expression or aspiration, but, instead, it should be repeatedly washed out with water. The quantity of the stomach-contents may also be increased, in which event motor insufficiency is not always the cause, for it may also be referable to the presence of abnormal constituents, as, for example, mucus, gastric juice, bile, or transudates. These substances may be excluded in many cases, and the increase in the quantity of the stomach-contents may then be directly attributable to mechanical disturbances of the motor apparatus. The degree of the latter may be estimated by repeated measurements of the stomach-contents. If remnants of the last meal are constantly found in the stomach-contents in the morning, it is always proof that gastric motility is markedly impaired, and in order to determine the degree of this disturbance it is desirable to examine the stomach when fasting.

The following methods are employed to accurately estimate the food remnants of the stomach:—

(*a*) *Method of Jaworski.*[1]—The principle of this method is as follows: if a certain amount of liquid is present in the stomach and a measured portion of a solution of a known concentration is added to the liquid in the stomach, this solution will be diluted by the fluid in the stomach. Or, if a substance is dissolved in the fluid in the stomach and a measured quantity of distilled water is introduced, the former will be diluted according to the quantity of the added water; and from the degree of this dilution a conclusion may be drawn as to the amount of liquid originally present in the stomach.

The symbols employed in the formulæ given below are defined as follows:—

x, The unknown quantity of liquid in the stomach (expressed in cubic centimetres).

y, The total quantity of substance dissolved in x.

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[1] W. Jaworski, Zeitschrift für Biologie, 1883, Bd. 18, S. 427-445.

p_1, The percentage of this substance in the liquid x (to be estimated after aspiration of a portion of this solution).

M, The amount of the definite solution which is introduced (expressed in cubic centimetres).

P, The total amount of substance dissolved in M.

p_2, The percentage of this substance after the liquid in the stomach has been mixed with the liquid M introduced into that organ, that is, the percentage of $x + M$ which is estimated after a second aspiration.

We have then:—

$$x : 100 = y : p_1$$

or

$$y = x \frac{p_1}{100}$$

furthermore,

$$(x + M) : 100 = (y + P) : p_2;$$

If y is substituted in this equation by its above value we have:

$$(x + M) : 100 = \left(x \frac{p_1}{100} + P\right) : p_2,$$

and therefore

$$p_2 (x + M) = 100 \left(x \frac{p_1}{100} + P\right)$$

and therefore

$$x = \frac{100\ P - p_2 M}{p_2 - p_1} \quad \text{(Formula Ia).}$$

Or if the same quantity of the solution (100 cubic centimetres) is employed in all the tests, the equation is simpler:

$$x = 100 \frac{P - p_2}{p_2 - p_1} \quad \text{(Formula Ib).}$$

Both of these formulæ may be employed for calculating the result when the percentage p_1, for example, of NaCl, acid, or alkali in the liquid in the stomach is known, and when a solution M (but of higher percentage) of the same substance, that is NaCl, acid or alkali, is introduced.

It is more convenient to introduce a definite solution M of a substance (P), which is not present in the stomach (for example, soluble sulphates, phosphoric acid, or salts of iron), and to estimate the dilution after mixing and aspiration.

In that case $p_1 = 0$, and the above formulæ are changed to

$$x = 100 \frac{P}{p_2} - M \quad \text{(Formula IIa),}$$

or

$$x = 100 \left(\frac{P}{p_2} - 1\right) \quad \text{(Formula IIb).}$$

This procedure is even more simplified when any substance dissolved in the aspirated stomach-contents, such as NaCl, acid, or alkali, can be accurately estimated, and thereupon introducing a quantity M of distilled water. In that event $P = 0$, and the two formulæ assume the following forms:

$$x = \frac{p_2 - M}{p_1 - p_2} \quad \text{(Formula IIIa)},$$

or when $M = 100$, $x = 100 \dfrac{p_2}{p_1 - p_2}$ (Formula IIIb).

The methods described in these formulæ are executed as follows: if we wish to proceed according to formula I or III, introduce a tube (cleaned with distilled water) into the stomach and aspirate until no more liquid is obtained; the fluid is collected in a dry vessel. This fluid is then diluted from two to four times (as may be required) with distilled water; these are thoroughly mixed and their p_1 estimated. The accurately measured quantity M (with the known quantity of P) is then introduced with the same tube into the stomach and mixed with the stomach-contents; the p_2 of this mixture $(x + M)$ is next estimated. The value of x is then easily calculated. If, on the other hand, it is desired to employ procedures described by the formula IIa and IIb, it will not be necessary to estimate p_1 and first aspiration is not needed. Soluble sulphates are most suitable for testing fluids, according to Jaworski.

This method was first employed by Jaworski for physiological and later[2] also for clinical purposes. This method, which possesses the advantage of being independent of acids in the stomach, has unfortunately not received popular recognition.

(*b*) *Method of Mathieu and Rémond.*[3]—The total quantity of stomach-content is more accurately determined by means of the method recently proposed by Mathieu and Rémond: a portion of the stomach-content is at first extracted and set aside. A definite quantity of water is then introduced into the stomach and allowed to thoroughly mix with the stomach-content by alternately elevating and lowering the funnel; as much of the stomach-content as can be obtained is then removed and collected in a special vessel. Now b represents the quantity of undiluted portion of the stomach-content which had been removed, a the acidity of it, a′ equals the acidity of the diluted stomach-contents, and q the quantity of the liquid introduced into the stomach; the acidities (a and a′) must be indirectly proportionate to the quantities, because the greater the quantity of the lavage water, the smaller the total acidity of the combined liquids in the stomach.

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[2] Jaworski, Wiener medicinische Wochenschrift, 1883, No. 12-13.

[3] Mathieu et Rémond, Soc. de biolog., 8 Nov., 1890, und Archiv für Verdauungskrankheiten, 1896, Bd. 1, S. 345.

Accordingly the following formula is formed:—

$$a : a' = q + x : x,$$

which gives
$$x = \frac{a' q}{a - a'}.$$

The quantity of the liquid originally in the stomach or the total amount retained equals then

$$b + \frac{a' q}{a - a'}.$$

The absolute amount of hydrochloric acid in the stomach at the time of aspiration can also be estimated with the help of these formulæ (see below).

The formula of Mathieu and Rémond assumes that the stomach-contents are acid, for it is not applicable in diseases of the stomach in which acid is either absent or so slight that, in mixing, the differences are no greater than the error of titration. To overcome this objection, P. Cohnheim[4] worked out a method based likewise on the principle of dilution, which can be used when acid is present or absent.

This method depends upon the following principles. When definite quantities of several acid solutions are mixed, the resulting acidity equals the sum of the products obtained by multiplying these quantities by the acidity, divided by the sum of the quantity expressed in cubic centimetres. For example:—

10 cubic centimetres with a total acidity of 40
10 cubic centimetres with a total acidity of 60
give an acidity of

$$\frac{(10 \times 40) + (10 \times 60)}{10 + 10} = \frac{400 + 600}{20} = 50$$

or, expressed arithmetically,

$$\frac{a \cdot A_1 + b \cdot A_2}{a + b}$$

In this formula A_1 and A_2 represent the total acidity, a the number of cubic centimetres of the first and b the number of cubic centimetres of the second acid solution. If now in place of the ordinary water used in the Mathieu-Rémond method of estimating the retained stomach-content, a definite amount (q) of a solution of hydrochloric acid is employed for mixing, the quantity of the retained contents can be determined even though hydrochloric acid is absent; in that case $a = q$. The total acidity is then:

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[4] P. Cohnheim, Centralblatt für innere Medicin, 1900, S. 441.

$$A_3 = \frac{x \cdot A_1 + q\,A_2}{x + q}$$

$$A_3\,(x + q) = A_1\,x + A_2\,q$$

$$x = \frac{q\,(A_2 - A_3)}{A_3 - A_1}$$

The total amount of retained stomach-contents is then equal to that remaining in the stomach plus that which has been expressed, that is,

$$G - R = a + X.$$

In order to make this calculation clearer we give (according to Cohnheim) the following example: one hour after a test-breakfast has been taken, fifty cubic centimetres of the stomach-contents are expressed (a), it having an acidity of 8 (A_1), for lavage, 400 cubic centimetres (q) of a solution of hydrochloric acid whose acidity (A_2) equals 65 is employed; the acidity of the mixture (A_3) equals 48. According to the above formula we then have

$$x = \frac{q\,(A_2 - A_3)}{A_3 - A_1}$$

In the case just described, we would then get

$$x = \frac{400 \times 17}{40} = 170$$

The total amount of retained stomach-content equals $a + x$, that is, $50 + 170 = 220$ cubic centimetres.

The old method of Mathieu and Rémond is recommended to estimate the quantity of retained stomach-content when acids are present; but if absent, this procedure should be substituted by that of Jaworski or Cohnheim.

(*c*) Similar to the above is the *method of Strauss.*[5] This author first determines the specific gravity of the undiluted stomach-contents, and then that of the latter when diluted with a known quantity of water. If S is the specific gravity of the undiluted, S′ the specific gravity of the diluted stomach-contents, V the quantity of stomach-content removed, and a the water added, then the quantity of retained stomach-content equals

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[5] Strauss, Therapeutische Monatshefte, 1895, Märzheft.

$$x = \frac{VS + (a - V) \times S' - a}{S - S'}.$$

If it is desired to determine only the quantity of fluid remaining in the stomach, the formula of E. Reichmann[6] is recommended:—

$$x = \frac{a\,(S' - 1)}{S - S'},$$

in which the letters have the same significance as in the formula of Strauss.

According to the method of Mathieu, about 150 cubic centimetres of stomach-contents are retained normally one hour after a test-breakfast; if this is essentially increased, a weakness of the motor activity may be suspected.

I have repeatedly made the observation that the results of the Mathieu-Rémond method do not coincide with those obtained with the Leube-Riegel test-meal (an empty stomach seven hours later). I have found very frequently that the Mathieu-Rémond method showed the presence of greater quantities of retained stomach-contents than those given above, whereas the Leube test-meal presented normal figures. I have never seen these conditions reversed.

In the method of Sörensen and Brandenburg,[7] which has been less generally adopted, a 30 per cent. solution of protogen (obtained by the action of formaldehyde upon the white of egg) is employed. The patient, on an empty stomach, takes 300 to 500 cubic centimetres of this solution, and from a half to an hour later the stomach-contents are expressed, immediately after which the retained stomach-contents are diluted with 100 to 200 cubic centimetres of water, which is then also expressed. The amount of nitrogen present is then estimated in 5 cubic centimetres of both solutions according to the Kjeldahl method. The amount of retained stomach-contents is then determined by means of a simple formula. The assistance of a large laboratory is needed for this method.

The *odour* of the stomach-contents is quite characteristic in many cases. It may be pungent-sour on the one hand, or, on the other hand, when acids are absent or an alkaline reaction exists it may be flat. When a large amount of blood is present, the stomach-contents have a peculiar sweetish odour similar to that of blood.

If the reaction is acid, the simple, distinct, sour odour which is caused by hydrochloric acid can be distinguished from that attributable to the fatty acids. The latter is, however, only demonstrable, at least after a test-breakfast, when extensive fermentative processes exist in the stomach. The odour of H_2S is also very characteristic and will be discussed later. The stomach-contents are fæculent when an abnormal communication exists between the stomach and the small intestines and especially the large intestines; the contents are fœtid in ulcerating carcinoma, phlegmonous gastritis, and in gastric abscesses.

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[6] E. Reichmann, Deutsche medicinische Wochenschrift, 1895; Litteraturbeilage, No. 12.

[7] Sörensen and Brandenburg, Archiv für Verdauungskrankheiten, 1898, Bd. 3, S. 377.

The odour of the stomach-contents is of pathognomonic value in the different forms of poisoning, for instance, garlic odour in phosphorus poisoning, the odour of bitter almonds in nitrobenzol poisoning, the well-known carbolic odour in carbolic acid poisoning. The odour of uræmic vomitus is ammoniacal.

It should be remarked, however, that the odour of the stomach-contents is not always due to gastric or intestinal disease, for it may also be referable to ulcerative processes of the oral cavity, the pharynx, or the œsophagus. Thus, as I have repeatedly observed, the stomach-contents may possess a fœtid odour in ulcerative stomatitis, in abscesses or cancer of the pharynx, and also in œsophageal cancer, abscesses of the antrum Highmori, etc.

Chemical Examination of the Gastric Contents.

Chemical examination of the gastric contents is undertaken with the object in view of obtaining, in the fullest sense of the term, a picture of the course of digestion in the stomach as well as of the processes to be completed in the intestines, and, by comparing it with the normal physiological functions, to gain information as to the existence of any *functional* disturbances or changes, and to secure useful information for the therapy.

Several points of significance in the methods of examination may be appropriately mentioned here.

A diagnosis as to the condition of the gastric functions can be established from a single examination only in the rarest cases (for example, in pronounced ectasia). It must be noted in this connection that fear of the stomach-tube does not uncommonly have an unfavourable effect upon the temperament which, as we know, can influence to a large extent the digestive processes. This holds especially true for neurasthenics, but is also applicable to healthy but timid individuals. I am of the conviction that in doubtful cases it is necessary, as a rule, to pass the stomach-tube three times, after as many test-meals, in order to become thoroughly informed as to the functional activity of the stomach; this does not include the passage of the stomach-tube when the stomach is fasting, which should be done, if at all possible, in every case.*

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* I wish to state here that the examination of the stomach with the tube should be dispensed with if possible during menstruation, for the researches of von Kretschy, Ewald, and myself and Elsner have shown that secretion of the gastric juice may be temporarily changed at this period (see above, page 104).

I have found from the abundant material in my private and hospital practice that after a primary and successful passage of the stomach-tube, the patient is quite willing to submit to a second examination. On the other hand, a patient can rarely be induced to agree to a second examination when the first exploration with a tube was for any reason *unsuccessful*. The physician must therefore exercise the greatest care and patience the first time a tube is passed.

Reaction of the Stomach-Contents.

After examining the stomach-contents macroscopically, a portion of it is filtered as carefully as possible (grooved filter) and tested for its reaction with litmus paper. The reaction may be acid, amphoteric, neutral, or alkaline. If the reaction has been found to be acid, the presence of *free* acids should be tested for.

We distinguish now for the first time between *free* acids and combined acids. If a very dilute solution of hydrochloric acid is added to one of albumen, the mixture will give an acid reaction from the moment that the hydrochloric acid is added; but not until after further addition is it possible to detect hydrochloric acid in the mixture, by means of the reactions about to be mentioned. Even though the hydrochloric acid which has combined with the albumen be latent for colouring substances, it is not without use to digestion, especially when combined with pepsine. There is no doubt nowadays but that digestion may proceed without *free* hydrochloric acid; it is simply less complete in comparison with that which occurs when free hydrochloric acid is present. It is as incorrect to assume, as is done by Salkowski and v. Jaksch, that the uncombined hydrochloric acid alone is capable of physiological activity, as it is to consider it as superfluous, as claimed by Martius and Lüttke according to Rosenheim's suggestion. If such were the case, the absence of free hydrochloric acid would necessarily be the rule and the presence the exception, whereas, as is well-known, the reverse is true. In my opinion, then, the terms "free" and "combined" hydrochloric acid are still the most appropriate. The albuminous bodies may be only slightly combined or saturated with the acid. If, for example, a dilute solution of hydrochloric acid or lactic acid is added drop by drop to a solution of albumen, the mixture will show acid reaction soon after the first drops; nevertheless the albumen is capable of absorbing a considerably larger amount of acid without allowing any of the acid to remain free. The albumen becomes saturated with acid when, upon the addition of the next drop of acid, it may be demonstrated by means of a colouring substance. The quantity of acid necessary to saturate the albumen may be designated as the "capacity for saturation." The latter varies not only with the different forms of proteids, but also, as was shown by the researches of F. Blum,[8] with certain modifications of albumen (syntonine, propeptone, peptone) which arise during digestion. It equals 80 to 90 per cent. (that is, 0.29 per cent. absolute or 1.16 officinal hydrochloric acid) in milk when a decinormal solution of hydrochloric acid is employed. Likewise, according to v. Pfungen,[9] the saturation capacity for meat equals 0.843 per cent. absolute or 3.3728 officinal hydrochloric acid. In other words, when a piece of meat is to be digested in a stomach which is not secreting hydrochloric acid, it would require at least 68 drops of our officinal hydrochloric acid for every 100 grammes of meat. The amount of hydrochloric acid required for such a small meal of meat (as 100 grammes) is in reality greater, for a portion of the acid which is introduced is lost by combining with the alkalies, salts, and albuminoid substances (mucin, mucus, cells, etc.) in the stomach. Even a roll (35 grammes) requires for its digestion, according to v. Pfungen, 0.944 gramme, and therefore, according to the above-mentioned example, a portion greater than 1 gramme of hydrochloric acid.

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[8] Blum, Zeitschrift für klinische Medicin, 1892, Bd. 21, S. 558-572.

[9] v. Pfungen, Wiener klinische Wochenschrift, 1889, No. 6-10.

The marked saturation capacity of albumen has also been proven by F. Moritz[10] through researches on the healthy human being. He found hydrochloric acid present in the free state not earlier than about four hours after a meal of meat, whereas after a meal of starchy food (potatoes) it was found in large quantities during the second hour.

The most sensitive test for free hydrochloric acid is furnished by means of **congo red.** It was discovered by Bötticher in 1884, and is obtained by the action of a molecule of the hydrochlorate of tetraazodiphenyl (from benzidin) upon two molecules of naphtionic acid. It was introduced into practice by v. Hösslin[11] and Riegel,[12] who recommended it in the form of congo paper as a reagent for free hydrochloric acid. According to Leo's[13] opinion, with which I agree, congo red in solution is even more sensitive than the congo paper. This solution is about ten times as sensitive as the paper, for by means of it 0.0009 per cent. of hydrochloric acid can be detected, whereas the paper does not react until 0.01 per cent. of the acid is present. Control experiments instituted by myself,[14] which have been essentially confirmed by Schäffer,[15] showed that in low percentages of hydrochloric acid, as, for example, 0.1 per mille and more, this colour reaction differs in no respect from that obtained by organic acids. Thus, for example, the blue colouration caused by 0.1 per cent. of hydrochloric acid corresponds to that due to 0.3 per cent. of lactic acid.

This may lead to confusion in certain cases. To avoid any error, treat the blue-coloured strip of paper with ether, which does not discolourise the blue colouration of hydrochloric acid, but does discolourise that of lactic acid. Only when the blue colouration is marked (azure [sky] blue) we may conclude without this test that free hydrochloric acid is present. Since, however, we possess absolutely reliable hydrochloric acid reagents, it is more useful to employ either the congo paper or congo solution simply as a reagent for *free acids, in which respect it certainly exceeds in sensitiveness the majority of the known acid reagents.*

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[10] Moritz, Deutsches Archiv für klinische Medicin, 1889, Bd. 44, Heft 2 und 3.

[11] v. Hösslin, Münchener medicinische Wochenschrift, 1886, No. 6.

[12] Riegel, Deutsche medicinische Wochenschrift, 1886, No. 35.

[13] Leo, Diagnostik der Krankheiten der Verdauungsorgane, Berlin, 1890, S. 99.

[14] Boas, Deutsche medicinische Wochenschrift, 1887, No. 39.

[15] Schäffer, Centralblatt für klinische Medicin, 1888, S. 841.

After congo paper has proven the presence of *free* acids, the examination should be directed toward free inorganic acids. We will next discuss:

The Reactions With Free Hydrochloric Acid.

Two large groups of reagents may be distinguished: 1, reagents which are altered in a characteristic manner by acids in general and mineral acids in special, and 2, reagents which form a characteristic combination only with mineral acids. The colouring substances which have proven the most excellent for these purposes are particularly the rosanilins and azo-dyes.

I. *Reagents which are caused to undergo colour changes by free acids (especially mineral acids).*

The rosanilin (fuchsin) dyes which are of particular use include the methyl- and gentian-violet, and the "brilliant vert" of the French.

The azo-dyes include tropæolin 00, which is designated as "l'Orange poirrier No. 4" by the French, the above mentioned congo red, benzopurpurin 6B (v. Jaksch[16]), and dimethylamidoazobenzol. All of the mentioned dyes, the majority of which play an important role in dye-works, are highly sensitive to acids, which, as a rule, cause to a more or less extent characteristic changes in their colouring substances. Their diagnostic usefulness in testing hydrochloric acid is due to the fact that organic acids change their colour, as a rule, only when these acids are concentrated, which occurs rarely or never in the stomach.

The sensitiveness of the different dyes for acids varies markedly. The table on page 164 presents the sensitiveness of the most useful hydrochloric acid reagents.

Methyl-Violet.—This represents a variable amount of methylated rosanilins; the greater the number of methyl groups present, the bluer the colour; and the smaller the number, the redder the products obtained. Thus the bluest is the hexamethyl-pararosanilin; the reddest, the trimethyl-pararosanilin. Maly[17] and Laborde[18] were the first to recommend it for physiological purposes, while van den Velden[19] was the first to employ it in clinical diagnostics. These dyes combine with hydrochloric acid, and, though more weakly, also with organic acids; although these resulting products have not as yet been carefully studied, they are distinctly characterised by changes of colour. Methyl-violet turns sky-blue even when only traces of hydrochloric acid are present.

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[16] v. Jaksch, Klinische Diagnostik innerer Krankheiten, 1901, 5 Aufl., S. 202.

[17] Maly, Zeitschrift für physiologische Chemie., Bd. 1, S. 147.

[18] Laborde, Gazette Médecine de Paris, 1874, No. 32-34.

[19] van den Velden, Deutsches Archiv für klinische Medicin, 1879, Bd. 23, S. 369.

The reaction test may be best obtained in the following manner. A methyl-violet solution is prepared of such concentration that when in a test-tube it gives a distinct violet colour; this solution is divided into two equal parts, to one of which filtered stomach-juice is added. If hydrochloric acid be present, the methyl-violet will turn into a deep sky-blue colour. The test may also be performed with the same results by placing the solution in two watch-crystals and adding some filtered gastric juice to one of them. A marked colour change (Ewald and Boas[20]) is also obtained by adding the methyl-violet to the stomach-contents in such a manner that it will form a layer above it. If hydrochloric acid be present, a beautiful sky-blue zone is formed at the point of contact, which is clearly distinguished from the methyl-violet contained in the dropper.

Brilliant green (brilliant vert) has been particularly recommended by Lannois[21] and Lépine.[22] This reagent is described by Lannois as follows: *Brilliant green* is a bluish green dye which turns into a beautiful blue when diluted with much water. If two or three drops of this reagent are added to a 0.1875 per mille solution of hydrochloric acid, the mixture begins to turn green; if a larger amount of hydrochloric acid be present the mixture will assume a yellowish tint, which, as may be plainly seen, rapidly increases so that in a 1.5 per mille solution the yellow colour is perfectly distinct. Lactic acid, when present in a concentration of 3 per mille, produces a greenish colour, but never a yellow tint; hence the presence of lactic acid does not interfere with the yellow colour due to hydrochloric acid. Georges,[23] A. Mathieu,[24] and Bouveret[25] also recommend vert brilliant and prefer it to methyl-violet because the colour changes are more pronounced. Krukenberg, on the other hand, failed to get the brilliant green reaction in gastric juices which responded distinctly to the other hydrochloric acid reactions. Martius and Lüttke[26] consider this reagent altogether worthless.

The most important of the azo-dyes include:

Tropæolin 00.—This dye represents the sodium salt of phenylamido-azo-benzol-p-sulphonic acid and is obtained by the action of paradiazo-benzol-sulphonic acid upon diphenylamin. When dissolved in alcohol the yellowish-brown solution thus formed is turned into a deep red upon the addition of dilute hydrochloric acid, lactic or acetic acid, and upon the addition of a large quantity a ruby-red colour is obtained. As small amounts as 2 to 3 per mille of hydrochloric acid may be detected in this manner. As

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[20] Ewald and Boas, Virchow's Archiv, 1885, Bd. 101.

[21] Lannois, Revue de médecine, 1887, No. 5.

[22] Lépine, Soc. Méd. des hôpitaux de Paris, 28 January, 1887.

[23] Georges, Archives de médecine expérimentale, 1889, S. 718.

[24] A. Mathieu, Therapeutique des maladies de l'estomac et de l'intestin, Paris, 1893, S. 21; Traité des maladies de l'estomac et de l'intestin, Paris, 1901, S. 76.

[25] Bouveret, Traité des maladies de l'estomac, Paris, 1893, S. 83.

[26] Martius and Lüttke, Die Magensäure des Menschen, Stuttgart, 1892, S. 46.

I have shown,[27] the tropæolin test, when performed with the following modifications, is an absolutely reliable and unequivocal method* for *detecting hydrochloric acid.* Three to four drops of a saturated alcoholic solution of tropæolin are placed in a porcelain dish, the edges of the latter are tilted so as to spread the solution about, and then an equal amount of gastric juice is added; the dish is again shaken in order to mix the two solutions. If the dish is now gently heated over a small flame, beautiful lilac or blue stripes form at the edges which are absolutely characteristic of hydrochloric acid. *A similar colour reaction is not obtained by organic acids, no matter what the degree of concentration may be.*

Tropæolin paper, which is more convenient and simpler in application, is prepared by soaking strips of good Swedish filter paper for a long time in a saturated alcoholic solution of tropæolin and then drying them. Gastric juice which contains hydrochloric acid at first discolourizes the paper brown to a more or less marked degree; upon heating, the brown is changed to lilac or blue. This colouration is also obtained spontaneously when the paper is dried at the temperature of the room. Organic acids in concentrated forms may also produce a brown colour, which, however, disappears upon heating or by allowing it to lie undisturbed for a long time or by treating with ether; but the blue or lilac tints are, however, never obtained with these acids.

*The tropæolin paper is accordingly an excellent test for information to the physicians. Confirmation by other methods needs only be resorted to when this test gives a weak or negative reaction.***

Congo red, see page 157.

Benzopurpurin 6B is recommended by v. Jaksch.[28] According to that author, this dye can detect even 0.39 milligramme of hydrochloric acid in 6 cubic centimetres of water. Benzopurpurin is employed, like congo and tropæolin, in the form of strips of paper. It has not stood the test of time.

Töpfer[29] has recently advocated a 0.5 per cent. solution of *dimethyl-amidoazobenzol* as a reagent for free hydrochloric acid. Extremely minute quantities of free hydrochloric acid discolour the reagent distinctly red; and indeed this test is ten times as sensitive to solutions of hydrochloric acid as congo paper and phloroglucin-vanillin. According to the researches of Strauss,[30] this preparation reacts also to acid phosphates and moderately concentrated solutions of lactic acid, but according to Hári[31] this is true only of artificial mixtures and not of native stomach-contents. This subject will be discussed in greater detail in the description of Töpfer's method for estimating hydrochloric acid.

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* Georges, *l.c.*, considers tropæolin as belonging in the same class with the reagent of Günzburg and the resorcin test, and indeed reports cases in which the latter tests were negative and the former positive. In a wide experience I have failed to note similar cases.

** I have employed the tropæolin paper test for over fifteen years and am perfectly satisfied with it. The poor results of others may be attributed to defective tropæolin, or poorly coloured paper.

[27] Boas, Deutsche medicinische Wochenschrift, 1887, No. 39.

[28] v. Jaksch, Klinische Diagnostik innerer Krankheiten, 1901, 5 Aufl., S. 202.

[29] Töpfer, Zeitschrift für physiol. Chemie., Bd. 19, Heft 1.

Vegetable Dyes.—Uffelmann[32] has employed and recommended the wine pigment, and the colouring substances of the mallow and the whortleberry as reagents for the detection of hydrochloric acid. These have, however, obtained no foothold in practice.

II. *Reagents which are altered only by hydrochloric acid in a characteristic manner.*

1. **Phloroglucin-Vanillin** (Günzburg's reagent).[33]—This preparation, which is composed of

Phloroglucini	2.0
Vanillini	1.0
Alcoholis absoluti	30.0

is an extraordinarily sensitive and also highly reliable reagent. It is employed by pouring three drops of the reagent from a small pipette bottle into a porcelain dish containing an equal quantity of the filtered stomach-contents; the two solutions are then thoroughly mixed.

Upon *carefully* heating over a small flame, a pretty carmine-red tint is obtained, especially around the edges. This colour is due to extremely fine crystals, which develop even in a watery solution of a dilution of 0.01 per cent. Nothing but fine red streaks are seen in a dilution of 0.005 per cent., no reaction is obtained in greater dilutions. No organic acid of whatever degree of concentration produces this colour reaction. Very useful and convenient, in my experience, is the filter paper prepared with Günzburg's solution. A piece of this paper moistened with two or three drops of stomach-contents and then heated will present beautiful carmine-red areas which remain uninfluenced by ether. [Günzburg's reagent produces a very similar colour reaction with boracic acid (preserved foods!).]

2. **Resorcin** (Boas' reagent).[34]—This reagent is composed of

Resorcini resublimati	5.0
Sacchari albi	3.0
Alcoholis diluti	ad 100.0

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[30] Strauss, Deutsches Archiv für klinische Medicin, 1895, Bd. 56, Heft 1 u. 2.

[31] Hári, Archiv für Verdauungskrankheiten, 1896, Bd. 2, Heft 2 u. 3.

[32] Uffelmann, Zeitschrift für klinische Medicin, 1884, Bd. 8, S. 393.

[33] Günzburg, Centralblatt für klinische Medicin, 1887, No. 40.

[34] Boas, Centralblatt für klinische Medicin, 1888, No. 45.

If three to five drops of this preparation are added to five to six drops of stomach-contents and the mixture heated over a small flame until completely evaporated, a pretty rose to vermilion-red colour is obtained, which disappears gradually upon cooling; this colour reaction resembles closely that of phloroglucin-vanillin. *Organic acids do not call forth a similar reaction.* The same result is reached by immersing a strip of Swedish filter paper into the stomach-contents containing hydrochloric acid, adding one to two drops of the resorcin solution, and then slowly heating. A violet colour is at first produced, but upon further heating a brick-red tint is obtained which is not decolourised by the addition of ether. The resorcin test, as is now generally acknowledged, "is in all respects equal to the Günsburg reagent" (Martius and Lüttke[35]), but requires greater care in its use.

3. Alphanaphthol Test of Winkler.[36]—When a small quantity of a strongly diluted solution of hydrochloric acid, or of stomach-contents containing hydrochloric acid, is placed in a porcelain dish with several granules of grape-sugar, and several drops of a 5 per cent. alcoholic solution of alphanaphthol are added, a blue-violet zone, which rapidly turns dark as ink, will form near the end of evaporation after careful heating.

Too rapid heating will easily cause products due to burning, which will disguise the blue colour. It is advisable, therefore, to perform the experiment over a water-bath. Instead of adding the grape-sugar in substance, we may employ a solution of grape-sugar in the alcoholic solution of naphthol; 0.5 to 1 per cent. of grape-sugar is sufficient.

This reagent contains, according to Winkler,

Alphanaphthol	0.5
Sacchari uvici	0.1
Alcohol. absol.	10.0

Winkler states that this reaction is positive even when the percentage of anhydrous hydrochloric acid is as low as 0.04 per mille. It does not respond to organic acids.

The Practical Value of the Hydrochloric Acid Tests.

The large number of reagents for testing hydrochloric acid make it necessary to refer to the practical value of the different ones. To begin with, there can be no doubt that the true hydrochloric acid reagents are to be preferred to others because of their ability to detect minimal amounts of hydrochloric acid under all circumstances and conditions. Nevertheless, the so-called dyed papers, namely congo and tropæolin paper, as well as dimethylamidoazobenzol also furnish very useful tests, which with much practice and experience may be employed for approximately correct quantitative estimations. I am inclined, therefore, to always use first the dye test in every examination of the stomach-contents, which in doubtful cases is followed by the employment of the true hydrochloric acid reagents.

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[35] Martius and Lüttke, Die Magensäure des Menschen, Stuttgart, 1892.

[36] Winkler, Centralblatt für innere Medicin, 1897, No. 39.

The use of fuchsin and azo-dyes in testing hydrochloric acid was objected to for the reason that the colour reaction was prevented or disguised by albuminoid bodies, albuminates, salts, etc. The degree of this influence was carefully and minutely studied by many. On the other hand, it was believed that the true hydrochloric acid reagents (phloroglucin-vanillin, resorcin) had the advantage that they remain unaffected by peptone and albuminoid bodies. I believe I was the first to emphatically call attention to the fallacy of such a deduction.[37] The above-mentioned tests were made by adding variable quantities of albumen, salts, etc., to stomach-contents containing hydrochloric acid, and then testing for hydrochloric acid. In this manner *the amount of free hydrochloric acid was influenced, and not the reagent*, and the absence or presence of the reaction was indicative of nothing else than whether or not free hydrochloric acid still existed after the addition of albumen, salts, etc. As a matter of course, every hydrochloric acid reaction becomes weaker, and finally disappears in proportion to the amount of albuminates present. The sensitiveness of a hydrochloric acid test depends upon how much albumen or peptone a solution to be analysed can contain without lessening the clearness of the reaction. *The same results can likewise be obtained by a far simpler means, that is, by gradual neutralisation of stomach-contents containing hydrochloric acid by means of sodium or potassium hydroxide.*

The opinion is not rarely expressed in literature that *free* hydrochloric acid cannot be detected by means of the usual reagents under abnormal circumstances, on account of the presence of high percentages of albumen, peptone, and salts in the stomach, or because of similar factors. Nothing can be more incorrect! If the mentioned conditions existed hydrochloric acid in the free state would not be present, as may be obviously seen from the above discussion. Such conditions occur already under physiologic conditions, as, for example, one and a half hours after a mixed meal or two to three hours after a meal rich in albumen, or also ten to fifteen minutes after the ingestion of a test-breakfast. Our object in diagnosis, however, is altogether different. Our endeavour is to determine whether free hydrochloric acid is present and, if so, to what extent, after the ingestion of certain *food in definite amounts* (test-breakfast, test-meal), which is followed by free hydrochloric acid normally after the elapse of a definite time.

The following table, partially based upon the careful researches of Krukenberg,[38] shows the limits at which the various hydrochloric acid tests will react. The tests are arranged in the order of their sensitiveness:—

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[37] Boas, Centralblatt für klinische Medicin, 1888, No. 45.

[38] Krukenberg, Inaug.-Diss., Heidelberg, 1888.

Dimethylamidoazobenzol detects even	0.002	per mille hydrochloric acid.
Congo solution	0.009	" " " "
Congo paper	0.02	" " " "
Alphanaphthol (according to Winkler)	0.04	" " " "
Phloroglucin- Vanillin Resorcin	0.05	" " " "
Methyl-violet	0.2	" " " "
Tropæolin 00	0.3	" " " "

Quantitative Estimation of Hydrochloric Acid.

All investigators have agreed that, as a rule, the amount of hydrochloric acid varies between 0.1 and 0.22 per cent., and therefore any *marked variation* from these figures must be looked upon as being abnormal. For that reason the quantitative estimation of hydrochloric acid is indispensable, even though it be possible with a little practice to determine the degree of acidity approximately by the colour reactions with hydrochloric acid. Before undertaking the quantitative determination of hydrochloric acid it is important to explain the elements to which the acid reaction of the stomach-contents is attributable. The following table presents the sources of this acidity:—

1. Hydrochloric acid
   - free
   - combined (with albuminoids, basic substances)
2. Organic acids (lactic, butyric, acetic acids)
   - free
   - combined (with albuminoids, basic substances)
3. Acid phosphates.

Either the total acidity of the stomach-contents may then be determined, in which case, if it is desired to reach any conclusions, the above-mentioned factors concerned in the total acidity must be taken into consideration; or we may estimate the total acidity due to hydrochloric acid (free and combined); or, finally, we may determine only the amount of free hydrochloric acid, or only the combined hydrochloric acid. The great amount of confusion which still exists on this subject requires, in my mind, the above classification of the methods for estimating hydrochloric acid. The question is also frequently discussed[39] whether the hydrochloric acid should be estimated in the filtered or unfiltered stomach-contents. It certainly would not be without practical value if we were able to determine the absolute amount of hydrochloric acid secreted from the stomach. This, however, is impossible, for one portion of the stomach-contents which contains hydrochloric acid has entered the intestines at the time of removal, another portion of the hydrochloric acid is absorbed and returned to the blood, a third portion is converted with neutral salts into acid salts, and a fourth portion is converted with organic bases into neutral salts; the amount of each of these components can be determined with difficulty or not at all. Finally, the exact estimation of a mixture which is so variable as the stomach-contents meets difficulties to which the exactness of the methods for the determination of hydrochloric acid is also subjected. The estimation of the relative amount (percentage) of hydrochloric acid in filtered stomach-contents is, under these circumstances, the simplest and most useful procedure.

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[39] Compare especially Geigel und Boas, Zeitschrift für klinische Medicin, 1892, Bd. 20, Heft 3.

1. Estimation of the Total Acidity.

The total acidity is determined by means of a decinormal solution of sodium hydroxide (see below) to which, as an indicator, is added, as a rule, phenolphthalein or litmus tincture.*

When phenolphthalein is used in the titration it is best to proceed by adding to ten cubic centimetres of the stomach-contents one to two drops of a 1 per cent. alcoholic solution of phenolphthalein (an otherwise indifferent substance), and then allowing as much decinormal soda solution to flow into it from a Mohr burette (shake the beaker containing the mixture well) as is required to give the fluid a faint reddish tint.

The calculation of the amount of hydrochloric acid is then based upon the *fact that one cubic centimetre of a decinormal solution corresponds to 0.00365 of hydrochloric acid.*

In this manner the total acidity, referable to the percentage of absolute hydrochloric acid, is easily determined. Thus an acidity of 5 (in 10 cubic centimetres) corresponds to 0.182 per cent. of hydrochloric acid.

We do not consider it superfluous to make a few explanatory statements on the preparation of normal solutions, for a physician is occasionally required to prepare them himself.

In the volumetric analysis we employ, besides the empyric volumetric fluids, compounded ad libitum, so-called *normal fluids, or normal solutions*, that is, fluids which contain the equivalent weight of the substance dissolved in the fluid.

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* The various indicators for the determination of hydrochloric acid are by no means equivalent. According to the researches of Lippmann (Diss.-inaug., Bonn, 1891) the percentage of hydrochloric acid is lower when rosolic acid is used than when litmus and phenolphthalein are employed. The same indicators should therefore always be employed, or the indicator which is selected should aways be mentioned in the report.

We speak accordingly of normal acid, normal alkaline, normal silver, and normal sodium chloride solutions, etc. When the normal solutions are diluted in a definite manner, as is necessary in order to attain the greatest degree of accuracy when titrating weak acids or, *vice versa*, weak alkalies, we speak of a fifth, a tenth, a twentieth, and a hundredth of a normal solution. Inasmuch as the equivalent weight of hydrochloric acid is 36.5 ($H = 1$, $Cl = 35.5$), and of sulphuric acid (H_2SO_4) 49 ($H_2 = 2$, $S = 32$, $4O = 64 = 98$; this is the molecular weight of sulphuric acid, but as it is dibasic, its equivalent weight would be the half of 98, or 49), a normal solution of hydrochloric acid would be such as contained 36.5 grammes hydrochloric acid to every litre, and a normal solution of sulphuric acid would contain 49 grammes of sulphuric acid to every litre. Now it is possible to obtain a normal alkali-solution, a cubic centimetre of which corresponds exactly to one cubic centimetre of the acid solution; and since the strength of a normal solution of hydrochloric acid or of sulphuric acid is known, the calculation becomes extraordinarily simple. If, for example, a litre of a normal solution of hydrochloric acid contains 36.5 grammes hydrochloric acid, each cubic centimetre of the accurately prepared alkali solution would correspond to 0.0365 hydrochloric acid. Since the equivalent weight of sodium hydroxide (NaHO) = 40, that of potassium hydroxide (KHO) = 56, therefore

1 c.c. normal soda solution................ = 0.04 NaHO,
1 c.c. normal potassium solution........... = 0.056 KHO, and
1 c.c. 1/10 normal soda solution........... = 0.004 NaHO,
1 c.c. 1/10 normal potassium solution...... = 0.0056 KHO.

Preparation of Normal Solutions.—It is best to employ oxalic acid for this purpose, the equivalent weight of which is 63 grammes. Accordingly, 63 grammes of oxalic acid corresponds to a litre of a normal alkaline solution, and 1 gramme of oxalic acid equals $\frac{1000}{63} = 15.87$ or, briefly, 15.9 cubic centimetres of a normal alkaline solution. Now dissolve 1 gramme of pure crystallised oxalic acid in a sufficient quantity of distilled water, add 2 to 3 drops of a solution of phenolphthalein, and titrate with diluted liquor potassæ, until a colour reaction is obtained. This test should be repeated two or three times. The number of cubic centimetres of liquor potassæ required to form a normal solution of caustic potash when added to a litre of water can then be readily calculated from the number of cubic centimetres of liquor potassæ employed. If, for example, we employed 12.5 cubic centimetres of caustic potash for 1 gramme oxalic acid, the following formula is obtained:

$$15.9 : 12.5 = 1000 : X$$
$$X = 786.1$$

that is, 786 cubic centimetres of the caustic potash to be employed is diluted to one litre. A decinormal baryta solution, etc., may be prepared in a similar manner. The following procedure is even simpler: 63 grammes of oxalic acid are dissolved in a litre of distilled water. Next dilute 150 grammes of liquor postassæ (Ph. Germ.) to 1050 and place the mixture in a burette; ten cubic centimetres of the normal oxalic acid solution is placed in a small beaker, and titrated with the prepared solution of caustic potash until the reaction becomes alkaline. As an indicator employ either phenolphthalein or rosolic acid. If now it is found that ten cubic centimetres of the acid are not neutralised by 10, but by 9.5 cubic centimetres of liquor potassæ, it is necessary to add to 9.5 liquor potassæ 0.5 cubic centimetre of water, or to 950, 50 cubic centimetres of water; then we possess a normal solution of caustic potash which is equivalent to the normal solution of oxalic acid, and of which every cubic centimetre corresponds exactly to 0.063 grammes of oxalic acid.

A normal solution of hydrochloric acid may be prepared according to the directions of the German Pharmacopœia. These are as follows: 146 grammes of hydrochloric acid having a specific gravity 1.124 (that is, officinal hydrochloric acid) are diluted with water to a litre. 1 gramme of the purest and freshly heated sodium carbonate requires for saturation 18.8 (more correctly 18.9) of this acid; for the equivalent weight of sodium carbonate is $53 = \frac{(Na_2CO_3)}{2}$; 1 gramme of sodium carbonate therefore equals $\frac{1000}{53} = 18.87$, or briefly, 18.9.

Of the normal alkaline solutions, the normal caustic potash and the normal baryta solution are to be preferred to the caustic soda, for the former attract less carbonic acid and therefore retain their titrating value longer. Normal solutions must be carefully preserved, in order that their titrating value will not be altered in the course of time. For the sake of accuracy the solution should be given a control test from time to time. The normal alkaline solutions tend readily to extract carbonic acid from the air, which, forming corresponding carbonates, have a not unimportant influence upon the titrating value of the fluid. It is perhaps superfluous to mention that burettes containing normal solutions must be well corked (rubber caps).

With reference to the technique, we wish to remind the examiner in reading the results obtained in titration, that the bottom of the meniscus is usually the point registered (the reading is made at the upper border of the meniscus when opaque fluids, such as solutions of iodine, potassium permanganate, etc., are used).

Reliable results can, to a certain extent, be obtained by direct titration with normal *alkaline* solutions in the above-described manner only when *pure gastric juice* is used. If, on the other hand, ingesta are present, even though they be as simple as that following a test-breakfast, the titration results offer but an approximate idea of the quantity of hydrochloric acid in the stomach-contents, for, aside from the latter, all other acids are included in the titration (organic acids, as well as acid salts formed during decomposition, especially phosphates). We must also remember that the *combined* hydrochloric acid is included with the *free* hydrochloric acid in the titration. The determination of the total acidity is wholly worthless when a large percentage of lactic acid is present (as, for example, in gastric cancer).

2. Estimation of the Total Amount of Hydrochloric Acid.

(a) According to Cahn and v. Mering.[40]

(α). *Titration Method.*—50 cubic centimetres of filtered stomach-contents are distilled over an open flame until three-fourths of it are distilled; then add a sufficient quantity to reach again 50 cubic centimetres and again distil until three-fourths are distilled. The *volatile acids* are retained in the distillate and are estimated by means of titration. The residue is well shaken in the same vessel at least six times with 500 cubic centimetres of ether each time; this causes the *lactic acid* to combine with the ether, and, after the latter has been evaporated, this acid is likewise estimated in the combined residue. The remaining acid residue is likewise titrated and the result is the amount of *free hydrochloric acid* present.

(β). *Cinchonine Method.*—The fluid, freed in the above manner of volatile fatty acids and lactic acid, is neutralised by the addition of an excess of cinchonine; the mass is treated with chloroform in a separating funnel; it is shaken with the chloroform four or five different times; the chloroform extracts are distilled, the residue dissolved in water, acidulated with a little nitric acid and the silver chloride precipitated by means of silver nitrate; this precipitate is weighed and the amount of hydrochloric acid is calculated from the result ($AgCl : HCl = 1 : 0{,}25427$).

The methods of Cahn and v. Mering detected, as was proven by the researches of Honigmann and v. Noorden[41] as well as Klemperer,[42] all of the various chlorine compounds occurring in the stomach, including also those combined with albumen; also the acid salts (phosphate, etc.) found in the stomach-contents upon titration, and finally, also, combinations of albumen with organic acids (lactic acid, butyric acid, albumen, etc.). The results of hydrochloric acid estimation are consequently not exact.

(b) According to Hehner-Seemann.[43]

This method was originally proposed by Hehner to detect whether acetic acid was adulterated by mineral acids, and later it was employed by Maly to examine the stomach-contents for hydrochloric acid. Seemann was the first to use this method for the estimation of hydrochloric acid. Identical with it is a procedure recommended by v. Leube[44] the origin of which he credits to A. Braun. When a definite quantity of a decinormal alkali solution is added to a quantity of vinegar, the mixture dried by evaporation, and reduced to ashes by slow, glowing heat, the organic acids become converted into CO_2 and disappear, thus permitting the easy calculation of the quantity of free hydrochloric acid from the amount of the alkali which remains behind. The gastric contents are treated in a similar manner.

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[40] Cahn and v. Mering, Deutsches Archiv für klinische Medicin, 1886, Bd. 39, S. 3 und 4.

[41] Honigmann and v. Noorden, Zeitschrift für klinische Medicin, 1888, Bd. 13, S. 87.

[42] Klemperer, Zeitschrift für klinische Medicin, 1888, Bd. 14, S. 156.

[43] Seemann, Zeitschrift für klinische Medicin, 1882, Bd. 5, S. 272.

[44] v. Leube, Spezielle Diagnose innerer Krankheiten, 1889, I, S. 234.

At first, determine the total acidity in a portion of the gastric filtrate (10 cubic centimetres) by titration with a decinormal alkali solution. Next, add to a second and equal portion of the stomach-contents a few cubic centimetres more than were necessary for neutralising the first portion. The alkaline solution is then carefully evaporated in a platinum or silver dish, or over an asbestos plate, and the residue reduced to ashes. The ashes are then dissolved in an equal number of cubic centimetres of a decinormal acid solution (hydrochloric acid or sulphuric acid); the solution is boiled in order to drive off the carbonic acid, and then titrated after phenolphthalein has been added as an indicator. The number of cubic centimetres of decinormal alkaline solution required for this purpose represents, when multiplied by 0.00365, the amount of hydrochloric acid in ten cubic centimetres. As may be noted, the quantity of organic acids in the stomach-contents may be determined in a very simple manner by means of this method, which is not true of any of the other methods.

The method is faulty as regards the fact that the acidity of the diphosphate salt is also taken into account, whereby the estimation of the value of the hydrochloric acid becomes too high. The correct figures, however, are obtained, according to Hári (Archiv für Verdaunngskrankheiten, 1896), in the final titration by means of dimethylamidoazobenzol as indicator. The method, according to Hári's exact investigations, is entirely faulty if free hydrochloric acid is absent; in such a case the final titration would merely express the presence of phosphates in the gastric contents.

(c) *After Sjöquist.*[45]

This procedure depends upon the principle that on the addition of carbonate of baryta the acids of the gastric juice are converted into the corresponding baryta salts. During the reduction to ashes which now follows, the baryta salts of the organic acids are converted into carbonate of baryta, while the $BaCl_2$ derived from the hydrochloric acid remains unaltered. The ashes are now extracted with hot water, the carbonates of bartya separating from the chloride of barium, only the latter going over. The amount of barium is now, according to the original directions of Sjöquist, determined by titration with a chromate solution, and from the barium chloride the HCl determined.

In detail, Sjöquist says as follows: Ten cubic centimetres of gastric contents in a silver or platinum dish with an excess of chlorine-free carbonate of baryta are evaporated to dryness with a gentle heat, the residue charred, and glowed for several minutes. After the cooling, the mass is mixed with 10 cubic centimetres of water, digested, and extracted repeatedly with hot water. The extract is filtered till the amount of the filtrate reaches 50 cubic centimetres. Then the baryta contained therein is titrated with potassium bichromate. To this end, the filtrate is mixed with one-fourth to one-third its volume of alcohol and with 3 and 4 cubic centimetres of a solution which contains 10 per cent. acetic acid and 10 per cent. sodium acetate. These additions are for the purpose of aiding the formation of the precipitate of baryta chromate, and, on the other hand, of preventing the formation of calcium chromate from the calcium salts possibly present, and of free hydrochloric acid. The end-reaction is detected by the use of the so-called tetra-paper (tetramethyl-paraphenyldiamin paper) which becomes bluish with the least excess of potassium chromate.

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[45] Sjöquist, Zeitschr. für physiol. Chemie, 1887, Bd. 13, Heft 1 and 2, P. 1.

Calculation:—For carrying out the titration, we need a solution of potassium bichromate of known strength, best 8.5 to 1000, the titrating value of which we must first determine, because the ordinary potassium bichromate is usually not pure. Each cubic centimetre of potassium bichromate employed till the beginning of the end-reaction corresponds to 4.05 milligrammes HCl. To determine directly the percentage of HCl in the gastric contents, we multiply the number of cubic centimetres of chromate solution that we have used with the number which represents 1 cubic centimetre and divide by the number of cubic centimetres of gastric contents used. For instance, if we have used 4 cubic centimetres of chromate solution, the percentage of $HCl = \frac{4 \times 0.405}{10} = 0.162$.

This titration method is rightly discarded on account of the unreliable end-reaction. Instead, Sjöquist[46] recommends another titration method. In detail, this is as follows: 10 cubic centimetres of gastric contents are mixed in a platinum or nickel dish with 0.5 thoroughly triturated barium carbonate, evaporated, and reduced to ashes. The ashes are repeatedly extracted with a little boiling water, and the filtrate (about 50 cubic centimetres) mixed with 4 cubic centimetres of ammonium acetate solution (produced by the neutralisation of 25 per cent. acetic acid and 10 per cent. ammonia) and one cubic centimetre of 25 per cent. acetic acid, boiled, and treated with 15 cubic centimetres of a 6 per cent. solution of neutral ammonium chromate. After two hours the precipitate is filtered out and washed free of chromate, and then dissolved with 10 cubic centimetres of water and a few drops of hydrochloric acid; 30 cubic centimetres of water, 2 cubic centimetres of iodide of potassium solution (50 to 100), and 5 cubic centimetres of a 25 per cent. HCl are now added. An amount of iodine exactly corresponding to the barium chloride is now set free, the determination of which yields the value of the HCl present in the gastric contents. This reaction occurs according to the following formula:—

$$2BaCrO_4 + 16HCl + 6KI = 2BaCl_2 + Cr_2Cl_6 + 8HO_2 + 6KCl + 3I_2.$$

The determination of the iodine follows in the known manner, by the use of hyposulphite solution (1 cubic centimetre should represent about 3 milligrammes HCl), with iodide of zinc starch as indicator.

Many modifications of Sjöquist's method have been offered:—

(a) *Weighing Method.*—v. Jaksch[47] has proposed to change the barium chloride into barium sulphate, to weigh the latter, and thus determine the amount of HCl in the gastric contents. Leo[48] and myself[49] have followed him, but the modification has not been adopted.

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[46] Sjöquist, Physiologische-chemische Bemerkungen über die Salzsäure, Leipzig, 1895.

[47] v. Jaksch, Klin. Diagnostik der innern Krankheiten, 1901, 5 Aufl., P. 207.

(β) *Modification of Bourget.*[50]—This requires two titration fluids: 1. A solution that contains exactly 1 per cent. of hydrochloric acid. 2. A soda solution 10 cubic centimetres of which exactly neutralise I cubic centimetre of the HCl solution. 10 to 30 cubic centimetres of the gastric contents are put in a small porcelain dish, and as much barium carbonate as will go on the end of a knife blade added. The fluid is evaporated and slowly reduced to ashes. Then we proceed exactly according to Sjöquist. The liquid containing barium chloride is treated with a concentrated soda solution (1 : 3); the precipitate of barium carbonate is collected, and washed until the washings no longer give an alkaline reaction. Filter and precipitate are now placed in a retort of 100 cubic centimetres capacity and 10 cubic centimetres of the titrated hydrochloric acid (1:100) spread thereon. We then shake lightly and fill the retort to the mark. The thoroughly well-shaken liquid is filtered, and 10 cubic centimetres of the filtrate in a small dish titrated with the above-mentioned soda solution, with phenolphthalein as indicator. The number of cubic centimetres of soda solution necessary for the neutralisation of the remaining acid gives directly the HCl amount, and a simple multiplication informs us of the total HCl contents.

(γ) As I have shown, we can simplify the method as follows:[51] After the precipitate of barium carbonate has been washed enough, we put it into a beaker containing an indefinite amount of water, separate filter and precipitate, and let a decinormal acid flow in until the collected carbonate of barium is in solution, and this reacts acid with litmus; after boiling (to drive off the CO_2), a little phenolphthalein is added, and the fluid is titrated with decinormal lye solution.

For instance: we have used 12 cubic centimeters of decinormal HCl solution, and at the titration with decinormal lye 7.5 cubic centimetres are used to neutralise; there are contained in the experimental fluid (10 cubic centimetres) $12 - 7.5 = 4.5$ decinormal hydrochloric acid, or in 100 cubic centimetres $= 45$ decinormal hydrochloric acid $= 0.164$ HCl.

Through comparison of the results of the titration and the weighing methods I have determined the practicability of the former, which possesses the advantages over the weighing method of simplicity and of requiring less time.

Favizky[52] proceeds in similar manner. According to this author, the barium chloride in solution is changed to barium carbonate. This is washed, dissolved in hydrochloric acid, evaporated to dryness, the residue dissolved in water, and titrated with weak silver solution. The amount of silver solution expresses directly the hydrochloric acid present in the gastric juice.

(δ) *Modification of Katz.*[53]—The extract obtained by washing the ashes with hot water is treated with several drops of a sal ammoniac solution, and made distinctly alkaline with ammonia. To this is added from a burette a titrating solution of potassium chromate until, with the employment of a freshly prepared solution of lead acetate (plumb. acetic, basic sol. 1:15) treated with ammonia, a distinct flesh-coloured precipitate occurs. This modification has found no application.

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[48] Leo, *l.c.*, S. 112.

[49] Boas, Centralblatt für klinische Medicin, 1891, No. 2.

[50] Bourget, Archive de médicine expérimentale, 1886, No. 6, P. 844 *et seq.*

[51] Boas, *l.c.*

[52] Favizky, Virchow's Archiv, Bd. 123, S. 292-300.

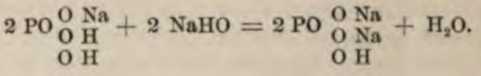
[53] H. Katz, Wiener medicinische Wochenschrift, 1890, No. 51.

Errors in Sjöquist's method have been pointed out by various authors (Leo,[54] v. Pfungen,[55] Martius and Lüttke[56]). Yet Sjöquist in his very detailed article, replete with numerous analyses, has shown that the said errors are partly avoidable (decomposition of sodium chloride) by painstaking technique, and partly not to be taken into account (ammonia). Sjöquist himself sees in his method only a means for the strictly exact estimation of the amount of hydrochloric acid; for practical purposes, however, it surpasses the ordinary chemical skill of the majority of physicians.

(d) After Leo.[57]

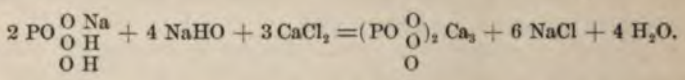
The method depends upon the fact that carbonate of calcium does not essentially influence a solution of potassium or sodium acid phosphate, while it fully neutralises the free acid in the cold. A sample of the gastric contents is mixed with as much powdered $CaCo_3$ as will go on the point of a knife, and if the previous acid reaction of the contents has disappeared, the latter contains only free acid and no acid salts; if the acid reaction remains as intense as previously, only acid salts are present; if it is slighter, both free acid and acid salts are present.

If the liquid contains both acids and monophosphates (which is mostly the case in the gastric contents), by agitation with calcium carbonate a loss of acidity is brought about, which corresponds to the acid present. If any soluble calcium salts (for example $CaCl_2$) be present, the monophosphates of potassium and sodium are converted into the corresponding calcium combinations. The presence of monocalcium phosphate needs, however, as the following reaction formulæ show, double as much lye for neutralisation as the corresponding amount of potassium or sodium phosphate.



$$2\,\mathrm{PO}\begin{matrix}\mathrm{O\,Na}\\ \mathrm{O\,H}\\ \mathrm{O\,H}\end{matrix} + 2\,\mathrm{NaHO} = 2\,\mathrm{PO}\begin{matrix}\mathrm{O\,Na}\\ \mathrm{O\,Na}\\ \mathrm{O\,H}\end{matrix} + \mathrm{H_2O}.$$

Further,



$$2\,\mathrm{PO}\begin{matrix}\mathrm{O\,Na}\\ \mathrm{O\,H}\\ \mathrm{O\,H}\end{matrix} + 4\,\mathrm{NaHO} + 3\,\mathrm{CaCl_2} = (\mathrm{PO}\begin{matrix}\mathrm{O}\\ \mathrm{O}\\ \mathrm{O}\end{matrix})_2\,\mathrm{Ca_3} + 6\,\mathrm{NaCl} + 4\,\mathrm{H_2O}.$$

On this account the number of cubic centimetres of lye at the second titration for the phosphates present must be divided by two. This division by two is not necessary when the first and second titration are carried out under the same conditions, namely: that also in the determination of the total acidity of a given example, calcium chloride in excess is added.

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[54] Leo, Deutsche medicinische Wochenschrift, 1891, No. 41.

[55] v. Pfungen, Zeitschrift für klin. Medicin, 1891, Bd. 19, Supplement Bd., P. 224-239.

[56] Martius and Lüttke, *l.c.*

[57] Leo, Centralblatt für die medicinischen Wissenschaften, 1889, No. 26. Consult also Leo, Diagnostik der Krankheiten der Bauchorgane, Berlin, 1895, 2 Aufl.

In detail, the HCl test is stated according to Leo's directions thus:—

Ten cubic centimetres of the filtered gastric contents are to be mixed with 5 cubic centimetres of a concentrated calcium chloride solution and titrated with decinormal lye. A second sample of the filtered contents is mixed with several grammes of a powdered calcium carbonate and filtered. Ten cubic centimetres of the filtrate (representing 10 cubic centimetres of the contents) are measured out and air is conducted through them for the purpose of driving off the carbonic acid, and, after 5 cubic centimetres $CaCl_2$ solution have been added, are again titrated with decinormal lye with the addition of a few drops of phenolphthalein as indicator. The difference between the first and the second titration represents the free acid contained in the gastric contents, that is to say, the hydrochloric acid, if, for example, fatty acids, such as lactic acid, have been previously removed.

This method has the advantage over the foregoing that on titration the possibly present phosphates remain out of account; moreover, this method accounts for not only the free acid but also the HCl combined with albumen and bases, because the acid albumin and also the muriatic albumoses, for the most part, unite with the calcium carbonate. It is, however, questionable whether, according to Leo's procedure, the collective organic acids are eliminated quantitatively.

Against the accuracy of the fundaments of this method a series of objections have been made by Hoffmann[58] (and Wagner) and in recent years by de Roig,[59] the first-named of which were rejected by Friedheim and Leo[60] under reference to the difference in experimental conditions. On the other hand, it follows from the work of the authors named that the method yields a correct result only by the use of absolutely chemically pure material and under the strictest adherence to Leo's directions.

(e) After Hayem and Winter.[61]

The principle of the procedure depends upon the fact that on the one hand the amount of total chlorine in the gastric contents, and on the other hand that amount of chlorine which is combined with metals, is estimated. The difference between the total chlorine and the chlorine of the chlorides represents the value for the total hydrochloric acid. Besides, the free hydrochloric acid is determined by drying at 100° C., at which temperature, according to the view of Winter, it should fully evaporate. Hayem and Winter state specifically as follows:—

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[58] A. Hoffmann, Centralblatt für klin. Medicin, 1890, No. 40.

[59] de Roig, Comptes rendus de la soc. de biol., 1899, S. 776.

[60] Friedheim and Leo, Pflüger's Archiv für die gesammte Physiologie, 1891, Bd. 48, S. 614.

[61] Hayem and Winter, Du chimisme stomacal, Paris, 1891, p. 72.

Five cubic centimetres of filtered stomach-contents are placed in each of three crucibles, *a*, *b*, and *c*; *a* is treated with an excess of sodium carbonate, and all three are dried in an incubator at 100° or in a water-bath. Then *a* is heated for some minutes to a weak red-glow with avoidance of any loss of substance until the sodium carbonate appears as a colourless enamel-like product. After cooling, this is treated with distilled water and a slight excess of nitric acid, is boiled to drive off the CO_2, and neutralised to a slight alkaline reaction with $CaCO_3$ or Na_2CO_3. After filtration and washing of the residue with boiling water, the filtrate is titrated with decinormal silver solution by means of potassium chromate as indicator. The figure found in *a* shows—expressed as to HCl—*the total chlorine contents of the gastric contents* (T = chlore totale).

The portion *b* is evaporated at 100° C. for one hour, then treated with an excess of sodium carbonate, again evaporated, and then handled as the first portion (*a*). The difference in the amount of chlorine between portion *a* and portion *b* signifies the volatile (H) chlorine, or, in other words, the *free hydrochloric acid* (HCl libre), namely, $a - b = H$.

The portion *c*, finally, after the drying, without further addition, is carefully reduced to ashes with the smallest possible flame and treated like *a*. The figure found expresses the value of the chlorides. $b - c$ represents the *chlorine combined with organic substances* (chlore combiné) = C.

Besides this, Hayem and Winter have worked out another value: $\frac{A-H}{C} = \alpha$. If we suppose the total acidity is represented only by HCl, then must $A = H + C$, or $\frac{A-H}{C} = 1$.[*] Now, as is known, organic acids occur very frequently in the stomach-contents. It follows that $\frac{A-H}{C}$ = not exactly 1, but > 1. There might also be a case in which $\frac{A-H}{C} < 1$; under such conditions an error of analysis would be present.

Hayem and Winter conclude, from the results obtained by means of the method just described, that the glands of the stomach do not secrete the finished hydrochloric acid, but chlorides; only, under the influence of albuminous bodies hydrochloric acid is set free. Upon these views Hayem builds an artificial structure of dyspeptic conditions. A discussion of this view is entirely superfluous, particularly as it stands in contradiction to all the experiences of the last decades.

The chief objection which has been made to the method depends upon the determination of H. According to the control experiments of A. F. Hoffmann,[62] Wagner,[63] Mintz,[64] Martius and Lüttke,[65] and Sansoni,[66] the value of b turns out too high because not all the free hydrochloric acid is evaporated on drying, and consequently the difference $a - b$, that is, the value for the free hydrochloric acid (H), is too low. As, furthermore, the combined hydrochloric acid (chlore combiné) is found out by the difference $b - c$, it is to be seen that the value for the combined hydrochloric acid is too high. Accurate only is the value for the total hydrochloric acid (a — c). Kossler[67] has further demonstrated that when a calcium chloride solution is mixed with a diphosphate and heated, a precipitate of monophosphate or normal calcium phosphate occurs; hydrochloric acid is at the same time set free, and escapes on evaporation. Consequently less chlorine is to be found in the residue than was actually present in the gastric contents.

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[*] Hayem and Winter give this value (α) as 0.86, but remark elsewhere that it approximates 1.

[62] A. F. Hoffmann, Schmidt's Jahrbücher, 1892, Bd. 223, S. 268.

[63] Wagner, Arch. de Physiologie norm. et path., Bd. 23, S. 440.

[64] Mintz, Deutsche medicinische Wochenschrift, 1891, No. 52.

[65] Martius and Lüttke, *l.c.*, S. 98.

[66] Sansoni, Berliner klin. Wochenschrift, 1892, No. 42 and 43.

(f) After Lüttke.[68]

Principle of the Method.—The normal stomach contains, as chlorine combinations, hydrochloric acid, potassium chloride, sodium chloride, and calcium chloride. On combustion of the contents only the hydrochloric acid, the free and also the organically bound, evaporates, while the chlorides decompose only at a strong red glow, and thus give off chlorine. If, then, in the gastric contents we determine, on the one hand, the total amount of chlorine, and on the other hand the amount which remains after combustion of the gastric contents, the difference between the two gives the chlorine which existed in the form of hydrochloric acid. The determination of chlorine is based on Vollhardt's determination of the halogens. Lüttke uses the unfiltered contents for the determination of the chlorine.

Details of the Method.—The following normal solutions are necessary:

1. *A Decinormal Silver Solution.*—17 grammes of silver nitrate to the litre, with an addition of ferrous sulphate as indicator. The solution is made thus: 17.5 grammes of silver nitrate are dissolved in about 900 cubic centimetres of nitric acid of 25 per cent. strength, and 50 cubic centimetres of liquor ferri sulphurici oxydati is added to the solution. The measure is filled to 1 litre. The standardisation of the solution is then made in the manner described for the measuring of the titration solutions for alkalies and acids, and that the most precisely when gauged by means of an accurate deci-hydrochloric acid solution.

2. *Decirhodanammonium Solution.*—7.6 grammes rhodanammonium contained in a litre. About 8 grammes rhodanammonium are to be dissolved in 1 litre of water and tested for the real amount of rhodan by means of the deci-silver solution. For this purpose 10 cubic centimetres of the exactly gauged deci-silver solution are placed in a beaker, diluted with water, and, under stirring, the rhodan solution to be tested is allowed to drop slowly from a burette until a permanent weak reddish colouration appears. If, for example, 9.7 cubic centimetres are used the solution is too strong, and 970 cubic centimetres of the rhodan solution must be diluted to 1000 cubic centimetres. The solution now made must be so conditioned that the first permanent red colouration appears exactly at the addition of 10 cubic centimetres.

Now follows first:—

(*a*) Estimation of the *total chlorine of the stomach-contents:* 10 cubic centimetres of the well-agitated stomach-contents are placed in a measuring retort of 100 cubic centimetres capacity. The little measuring retort is repeatedly rinsed with water, then 20 cubic centimetres of the deci-silver solution are added, then shaken and allowed to stand for about ten minutes.* Now the measuring retort is filled to 100 cubic centimetres, agitated, and the mass filtered through a dry filter into a dry beaker. 50 cubic centimetres of this are now titrated with deci-rhodan solution.

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[67] Kossler, Zeitschrift für physiol. Chemie, 1893, Bd. 17, S. 91.

[68] Lüttke, Deutsche medicinische Wochenschrift, 1891, No. 49.

The calculation of the total chlorine now follows in such a manner that the number of cubic centimetres of rhodan solution used must be multiplied by two, and this subtracted from the number of the amount (20 cubic centimetres) of silver solution employed.

(*b*) Estimation of *mineral chlorine:* 10 cubic centimetres of the uniformly agitated stomach-contents are carefully evaporated in a platinum, silver, or nickel dish upon a water-bath (asbestos plate, etc.). After this is fully dried, the residue is to be burned over a flame, but only until the mass no longer burns with illuminating flame. After the burning and the cooling, the ashes are to be extracted with 100 cubic centimetres water and filtered. If at the end of extraction we are in doubt as to whether all the chlorine is washed out, we test the last filtrate with a drop of silver nitrate. If cloudiness appears, chlorine is still present, and the washing must be continued.

The total filtrate is now mixed with 10 cubic centimetres decisilver solution and titrated with decirhodan solution. The estimation of the mineral chlorine is found by subtraction of the found number cubic centimetre of rhodan solution from the amount of silver (10 cubic centimetres) employed.

Estimation of Hydrochloric Acid.—The same is derived from the difference between the value of the total chlorine and that of the mineral chlorine, or, in other words, from the difference between the number of cubic centimetres determined for a and b.

Example:—

We have found for a = 6.5 cubic centimetres decinormal silver solution,
" " " " b = 2.3 cubic centimetres decinormal silver solution,

so that the hydrochloric acid content for 10 cubic centimetres = 4.2, or 1 cubic centimetre normal silver solution represents 1 cubic centimetre normal hydrochloric acid solution = 4.2 × 0.0365 = 0.153 per cent. hydrochloric acid.

The method of Lüttke is likewise not without errors; the first is an objection made also to Winter's method according to Kossler[69] and Sjöquist,[70] and consists of the fact that on heating of the chloride of calcium and double phosphates, hydrochloric acid is set free; the second error is due to the fact that, according to the experiments of Rosenheim,[71] Strauss,[72] and Honigmann,[73] ammonia is present in measurable quantities in the normal stomach-contents. The chloride of ammonium present decomposes on glowing, whereby, as was the case in the first-named error, the value of the HCl must be rated as too high.

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* Lüttke recommends, if the gastric contents are highly coloured, discolouration by the addition of a few drops of permanganate solution; but although I have made numerous estimations according to this method, I have never had occasion to use it.

[69] Kossler, *l.c.*

[70] Sjöquist, Physiologisch-chemische Beobachtungen über die Salzsäure, Leipzig, 1895. Consult also Zeitschrift für klin. Medicin, Bd. 32, S. 451.

[71] Rosenheim, Centralblatt für klin. Medicin, 1892, No. 39.

[72] Strauss, Berliner klin. Wochenschrift, 1892, No. 17.

[73] Honigmann, Berliner klin. Wochenschrift, 1893, No. 15 and 16.

(g) After Töpfer.[74]

This method depends upon the idea of determining the acid factors separately; that is, the total acidity, the free, and the combined hydrochloric acid. From the difference between the total acidity and the value for the free and the combined hydrochloric acid is derived the value for the organic acids and the acid phosphate. Specifically, the author works as follows:—

The free hydrochloric acid is estimated by means of dimethylamidoazobenzol in 0.5 per cent. solution. The yellow colour of the just-mentioned reagent changes to a reddish colour through the addition of the slightest amount of hydrochloric acid. *Organic acids give a similar colouration only in a concentration of over 0.5 per cent.,* and the presence of albuminous bodies requires a still greater concentration to produce the named colour alteration. One titrates, after the addition of a few drops of the reagent, with decinormal lye, until the reddish tone disappears and gives place to the original yellow.

For the estimation of the loosely combined HCl Töpfer uses alizarin (sodium alizarinsulphonate), which is said to be sensitive to all the acid factors except the combined HCl. After the addition of 3 to 4 drops of a 1 per cent. watery solution of alizarin, we titrate until the appearance of the first pure violet colouration.

The total acidity is estimated according to the usual procedure—titration with decinormal lye with phenolpthalein as indicator. The difference between the phenolphthalein value and the alizarin value yields the amount of the loosely combined hydrochloric acid.

Investigation by Einhorn,[75] Strauss,[76] and Hári[77] has shown that errors occur also in Töpfer's procedure. According to the last-named author, it is, however, a reliable method when free hydrochloric acid is present, and gives us, in the shortest time and in a very simple manner, as correct results as the new Sjöquist method (see p. 169) and the Braun (Hehner-Seemann) procedure (p. 168). When free hydrochloric acid is absent, Töpfer's method is adapted neither for the quantitative determination nor for the detection of the presence or absence of hydrochloric acid in general.

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[74] G. Töpfer, Zeitschrift für physiol. Chemie, 1894, Bd. 19, Heft 1.

[75] Einhorn, New York Medical Journal, 9 May, 1896.

[76] Strauss, Deutches Archiv für klin. Medicin, 1896, Bd. 56, Heft 1 u. 2.

[77] Hári, Archiv für Verdauungskrankheiten, 1896, Bd. 2, Heft 1 u. 2.

(h) *After v. Mierzynski.*[78]

v. Mierzynski's method is a gasvolumetric method. For its carrying out the well-known Wagner azotometer is necessary.

Five, ten and twenty cubic centimetres of the stomach-contents are placed in a porcelain crucible with an excess of barium carbonate, evaporated, and in the covered crucible glowed to a thorough carbonisation, and extracted repeatedly with water. According to v. Mierzynski some barium carbonate is frequently reduced through the carbon, and goes into solution as hydroxide, and is falsely reckoned as HCl. In order to avoid this, the filtrate is treated with phenolphthalein; if a red colour results, air is blown through to precipitate the hydroxide. The barium chloride solution is precipitated by monochromate of ammonium ($CrO_4 [NH_4]_2$) and filtered, and the precipitate washed out with dilute ammonia. The precipitate is now rinsed with hydrochloric acid (1:20) into the outer space of the generating vessel, and 10 cubic centimetres of diluted sulphuric acid are added. Water is brought to the same level in both limbs of the azotometer, measuring up to the mark O on the graduated tube, after which a little water is allowed to flow out of the not-graduated tube. The little vessel in the development-vessel is filled with 5 to 10 cubic centimetres of commercial hydrogen peroxide (2 to 2.5 per cent.). The generating vessel is closed, as well as the rubber tube; the hydrogen peroxide is mixed rapidly with the liquid containing chromate and the stopcock opened so that the oxygen enters the graduated tube. The stop is closed, and the vessel is then shaken sharply to and fro three to four times, lastly for five minutes. The generating vessel is now placed in water, and in about ten minutes the water niveau in both limbs of the azotometer is set at the same level, the volume of the oxygen generated, as well as the barometer and the temperature of the water, are read off, and the HCl calculated therefrom, whereby 1 atom $O = \frac{1}{2}$ molecule HCl. The read-off volume of O is reduced according to the table of Baumann,[79] and then multiplied with half the molecular weight of $HCl = 18.185$.

This method has been tested by H. Wiener[80] and found to correspond well with other methods. It is clear that it is not adapted for daily practice.

Similar to this method is the procedure of Riegler,[81] likewise gasvolumetric. It depends upon the Sjöquist method, except that the barium chloride is not titrated, but estimated gasvolumetrically. This method also is not adapted for practical purposes.

(i) *After v. Moracewski.*[82]

This method depends on the characteristic, that an ether-alcohol mixture takes up hydrochloric acid, but not salts. The hydrochloric acid in the mixture can be readily determined by titration.

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[78] Mierzynski, Centralblatt für innere Medicin, 1894, Bd. 15, S. 1073.

[79] Baumann, Zeitschrift für angew. Chemie, 1891.

[80] Wiener, Centralblatt für innere Medicin, 1895, No. 12.

[81] Riegler, Deutsche medicinische Wochenschrift, 1902, No. 25.

[82] Moracewski, Deutsche medicinische Wochenschrift, 1896, No. 2.

Procedure: Ten to fifty cubic centimetres of gastric contents are evaporated in a small dish to one cubic centimetre. This remainder is then put into a retort of a capacity of 100 cubic centimetres which in turn is filled to the mark with an alcohol-ether mixture (25 alcohol, 75 water-free ether). When the retort is filled to the mark, it is well shaken. After a short pause, 50 cubic centimetres of the solution are filtered off, put in a small retort which may be closed by a glass stop-cock and which has a capacity of 250 cubic centimetres.

To the 50 cubic centimetres ether-alcohol extract add about the same amount of water, and deci-normal soda solution in a quantity corresponding to the fourth part of the amount of gastric juice used in the work. To the neutral solution, which separates into two strata, add 1 to 3 drops of neutral potassium chromate, and titrate with deci-silver solution. The end point is shown by the red colouration of the silver chloride. Calculation is made in the usual manner.

This method appears rather simple and useful. It is, however, questionable whether at the evaporation a small amount of hydrochloric acid is not lost. No one has as yet substantiated the procedure:

(k) After Cordier.[83]

This method depends upon the principle that a mixture of equal parts of absolute alcohol and water-free ether takes from a mixture of sodium chloride and lithium chloride only the latter. Cordier places 5 cubic centimetres of gastric contents into a small dish with lithium carbonate until an alkaline reaction with litmus is shown; it is then evaporated on a water-bath, and charred at the lowest possible temperature.

The charred mass is extracted with alcohol-ether, and the filtered-off extract, which contains the chlorine of the free and loosely combined hydrochloric acid as lithium salt, is titrated, after the addition of an equal volume of water and a few drops of chromate of potassium, with decinormal silver nitrate solution. The charred residue is extracted with warm water, the extract boiled after the addition of a few drops of nitric acid, and filtered through the same filter that served for the first filtration, then washed with warm water. The united filtrates are made very weakly alkaline with sodium carbonate, and on titration as above the chlorine of the combined chlorides is determined. The amount of free hydrochloric acid is determined by titration with decinormal soda solution, with the use of phloroglucin-vanillin as indicator.

The method seems to be exact and is worthy of further tests.

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[83] Cordier, Compt. rend., Bd. 126, p. 353 to 356; refer also to Maly's Jahresbericht, 1898, p. 345.

3. Estimation of the Free Hydrochloric Acid.

(a) Method of Mintz.[84]

This method depends upon the fact that if the limits of a reagent for hydrochloric acid are exactly known, a conclusion as to the amount of HCl in the given stomach-contents can be drawn from the cessation of the reaction on titration by means of a decinormal alkaline solution. Günzsburg's reagent serves as indicator, and its reaction limit is 0.036 per mille HCl. If, for example, one finds that the reaction fails to appear after the addition of 1.3 decinormal lye, although with 1.2 it still remains positive, the free hydrochloric acid to be found in the test liquid calculated for 100 cubic centimetres is 12 + 1 decinormal lye = 13 cubic centimetres decinormal lye, or 0.047 per cent. HCl.

The method has been substantiated by many investigators. It has been generally accepted that the same, after some practice, yields a reliable value for the free hydrochloric acid.

Instead of Günzburg's reagent, one may employ, as Rosenheim[85] has discovered, filter paper which had been soaked in Günzburg's reagent and dried.

(b) Method of Mörner[86] *and Boas.*[87]

Independently of each other, Mörner and myself suggested a method to determine quantitatively the free hydrochloric acid of the gastric content by means of congo-red dye. Mörner gives a test-meal consisting of a soft-boiled egg, 30 grammes cakes [biscuits], and 250 cubic centimetres meat-broth. One hour later the test-meal is removed. Hereby the value of the combined hydrochloric acid remains almost constant at 0.05 per cent. and the free HCl is estimated by means of decinormal lye with congo red as indicator. The found value of hydrochloric acid + 0.05 per cent. gives the total HCl contents of the test-fluid.

I use exclusively congo red in watery solution,* 5 cubic centimetres of which are added to an equal amount of the test-fluid. Then I titrate with decinormal lye until the liquid becomes again distinctly brick-red. The number of cubic centimetres of normal lye employed gives directly the amount of free hydrochloric acid. The slight amount of organic acids that are titrated along with HCl hardly need be taken into account as regards exactness for ordinary practical purposes. Only, if large amounts of organic acid are present, it is necessary to rid the test-fluid from the same before titration, through repeated agitation with ether.

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* Alcoholic solution of congo produces a cloudiness with stomach-contents containing hydrochloric acid, that makes the titration result unreliable.

[84] S. Mintz, Wiener klinische Wochenschrift, 1889, No. 20.

[85] Rosenheim, Deutsche medicinische Wochenschrift, 1891, No. 49.

[86] Carl Th. Mörner, Upsala Läkareförenings Förhandlingar, Bd. 24, p. 483 and 491. After Maly's Jahresbericht für Thierchemie, 1889, Bd. 19, p. 253.

[87] Boas, Allgemeine Diagnostik und Therapie der Magenkrankheiten, 1890, 1 Aufl., p. 134, and Centralblatt für klinische Medicin, 1891, No. 2.

In similar manner, Martius and Lüttke have employed tropæolin as indicator, but the latter does not possess any advantage over congo-red.

Method of Hoffman.[88]

This clever method depends upon the fact that the hydrochloric acid converts cane-sugar into invert sugar, that is, equal parts of lævulose and dextrose, wherefore it stands to reason that the polarisation correspondingly changes. Organic acids have this power in such a slight degree, that the alterations which they produce are not to be considered for practical purposes. For qualitative and quantitative determination of HCl, five flasks, all alike, are needed. No. 1 is to hold a definite amount of cane-sugar and hydrochloric acid; No. 2 contains the same amount of cane-sugar and gastric juice; No. 3, only pure gastric juice; No. 4, gastric juice, cane-sugar, and sodium acetate (the last to observe, through the neutralisation of the hydrochloric acid, the possible influence of the ferment). The polarisation of all four samples is determined, then they are all placed in an incubator for several hours and again polarised. If HCl is present, the rotation is materially reduced in samples Nos. 1 and 2, while that of Nos. 3 and 4 are not materially altered. From the known amounts of HCl of No. 1 the HCl can be determined according to the formula: $\log A - \log (A - x) = C$, the absolute amount of HCl in the gastric juice.

Later, Hoffman simplified the above method in such a manner that, instead of inverting cane-sugar, he splits methyl-acetate into methyl-alcohol and acetic acid. After this splitting of the products, the acetic acid is determined by titration.

According to the investigations of Kossler[89] on an artificial mixture, and of Sjöquist, the method is exact in itself, and easily carried out. It is a pity that it has found such a limited use in practice.

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[88] Hoffman, Centralblatt für klinische Medicin, 1889, No. 46; consult also Schmidt's Jahrbuch, 1890, Bd. 255, p. 77.

[89] Kossler, *l.c.*

4. Estimation of the Free Hydrochloric Acid and the Total Acidity.

For this purpose, Citron[90] has recently contributed an easily used method, especially adapted for practical purposes. The author calls his instrument an acidimeter (Fig. 25).*

Fill the acidimeter-tube to the mark *M* with filtered gastric juice (if the amount is not sufficient, fill with water afterwards to mark *M*; the obtained figures are to be multiplied by two), add two drops of "reagent" (dimethylamidoazobenzol and phenolphthalein of each 1.0, alcohol 10.0), and add from a corresponding bottle or burette decinormal NaOH until the red or reddish-yellow colour of the liquid in the acidimeter begins to turn a distinct canary-yellow. After each addition, the rubber stopper is put on the tube, which is then to be inverted without shaking. When the yellow colour appears, one reads the level of the liquid on the red graduation of the right side and gets directly the percentage of the amount of HCl of the gastric contents. The obtained figure is noted, and then continue to add to the same liquid in like manner decinormal NaOH with steady inversion of the tube, until the liquid has become distinctly and *permanently red*. The total acidity is now determined by reading the level from the yellow graduation on the left side.

If in the beginning the gastric juice did not contain any free HCl, only a dirty yellow colour is obtained on addition of the "reagent." In this case the lye-solution must be added very cautiously, and continued until the red colour is reached, which marks the total acidity.

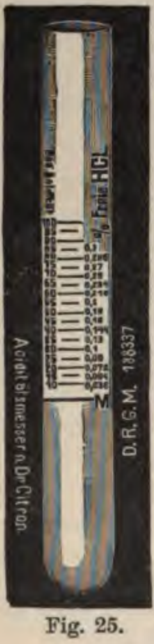

Fig. 25.

5. Estimation of the Combined Hydrochloric Acid.

The estimation of the combined hydrochloric acid has for its object the ascertaining how much hydrochloric acid is needed till the appearance of the known colour reaction, *i.e.*, until the formation of free hydrochloric acid.

Several methods are used for this purpose:—

(*a*) To a measured amount of filtrated stomach-contents, that much of decinormal hydrochloric acid is added from a burette until either the congo red or, better, the phloroglucin-vanillin or resorcin reaction appears. If 1 cubic centimetre of decinormal hydrochloric acid is subtracted for each 100 cubic centimetres of gastric juice, the remainder represents the amount of HCl which the stomach would have to supply for the formation of free hydrochloric acid. If now in a second portion, to which the found amount of hydrochloric acid has been added, the total acidity is determined, and if the value of hydrochloric acid which was necessary till the appearance of the phloroglucin-vanillin reaction, etc., is subtracted from the total acidity, the remainder represents the value of the *combined* hydrochloric acid. This procedure is also called the estimation of the hydrochloric acid deficit.

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*[The Valzahn Co., of Philadelphia, has put a similar instrument on the market, which has the advantage of having a glass stand. As frequently the amount of gastric contents is only small, I improved the instrument by graduating the lower part of the tube also, so that the contents may be easily and accurately diluted.]

[90] Citron, Verhandlungen des Vereins für innere Medicin, January 5, 1903.

J. Ehrmann[91] is somewhat more precise and defines the hydrochloric acid deficit as the difference between the amount of hydrochloric acid which might have been combined at all and that amount actually combined in the special case, and that based on the test-breakfast. As Ehrmann has estimated the amount of hydrochloric acid combinable in a test-breakfast as much as 0.6 to 0.7 decinormal HCl, he determines the deficit, in cases of absence of free hydrochloric acid, through addition of decinormal hydrochloric acid. Thereby he obtains the result that if a deficit of 0.05 to 0.07 per cent. has been found, no hydrochloric acid secretion has developed in the stomach.

It is evident that sources of errors are to be found in either method. It is especially evident in the method of Ehrmann, who depends upon the porridge, consisting of a roll mixed with water, while there is added through the act of mastication a considerable though varying amount of substances (mucin) which bind HCl. Furthermore, the "hydrochloric acid deficit" based on a test-breakfast is an absolute figure which does not admit of reference in percentage to stomach-contents containing varying amounts of water. The method is absolutely useless, as Ehrmann himself states, in cases in which many lactates are present, for a part of the added hydrochloric acid is used to decompose the same.

(*b*) *Method of O. Cohnheim and Krieger.*[92]—The principle of this method depends upon the fact that when a salt of phosphortungstic acid is added to an acid solution containing albumen hydrochlorate, an insoluble precipitate of phosphortungstic albuminate results. Hereby a loss of acidity occurs, which exactly expresses the amount of hydrochloric acid combined with albuminates.

The procedure in detail is as follows:—The total acidity and the free HCl are determined for ten cubic centimetres of the filtrated gastric contents. Other 10 cubic centimetres are treated with calcium phosphortungstate.* After filtration the acidity is again determined. The difference from the previously determined total acidity represents the combined hydrochloric acid.

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* Calcium phosphortungstate may be obtained as follows: A 4 per cent. solution of phosphortungstic acid while boiling is neutralised with calcium carbonate and filtered.

[91] J. Ehrmann, Berliner klin. Wochenschrift, 1897, No. 51.

[92] Otto Cohnheim and Krieger, Münchener med. Wochenschrift, 1900, No. 12.

When free hydrochloric acid is absent, the hydrochloric acid deficit is first determined, then a definite amount of hydrochloric acid (preferably 30 to 40 cubic centimetres more than the deficit amounts to) is added. The amount of the deficit is to be subtracted from the value of the combined hydrochloric acid.

(*c*) For more exact investigations one may also choose one of the above-described methods for the determination of the free and the combined hydrochloric acid (Hehner-Seemann, Sjöquist, Martius-Lüttke, Leo). That these methods also are not free from errors has already been mentioned.

Practical Value of the Various Estimation Methods for Hydrochloric Acid.

In the discussion of the value of the methods for estimation of hydrochloric acid, it must first be made clear that, as far as clinical purposes go, our only object is to recognise gross deviations from the normal chemical function. We cannot afford to take into account slight differences, because the physiological stomach, as the investigations of Rosenheim,[93] Verhaegen,[94] and others have shown, is subject to certain and by no means insignificant variations of secretion; and secondly, because we have to deal, not with an unalterable substance in the gastric contents, but with a material which, as to the amount of the water, the substances dissolved therein, abnormal by-products, etc., affords the most diverse variations. For this reason, it would perhaps be most appropriate for us not to estimate the quantity of hydrochloric acid at all, but to convince ourselves by means of colour tests (congo, tropæolin, dimethylamidoazobenzol, etc.), if at a certain stage, when HCl is usually to be found in a free state, whether it is present or not.

This may be sufficient for some, but not for all cases. Clinical experience shows that there are considerable deviations, both above and below the normal state, frequently accompanied by disorders which cease as soon as the secretion of the gastric juice becomes normal.

For this purpose, the recognition of the HCl amount is of great importance. For clinical purposes, in the majority of cases, we may content ourselves with the estimation of the total acidity, using the method of Mintz, or Mörner-Boas, or Töpfer for the determination of free hydrochloric acid. All other methods serve exclusively scientific purposes.

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[93] Rosenheim, Deutsche medicinische Wochenschrift, 1892, Nos. 13 and 14.

[94] Verhaegen, La Cellule, T. XIV, Fasc. 1; reference in Centralblatt für innere Medicin, 1899, p. 22.

Diagnostic Significance of the Hydrochloric Acid Test.

In the last decades, the qualitative test, and particularly the quantitative estimation, of hydrochloric acid in the gastric contents has come to play a prominent rôle in the diagnosis of the diseases of the stomach.

If we want to judge the same correctly, which is not always the case in recent literature, it is absolutely necessary to treat briefly the question of the source of the hydrochloric acid. Although little may be stated with certainty, it is evident that the secretion of this acid depends on three components: first, on the presence of the hydrochloric acid producers in the blood (Maly, Bunge, Forster, Cahn); then upon the integrity of the glandular apparatus of the stomach; and finally upon the nervous apparatus supplying the stomach. From this we may conclude, that defective and excessive secretion of hydrochloric acid may occur under three circumstances: first, during a morbid condition of the blood, which may have a favourable or injurious influence upon the production of HCl; second, during a disturbance of the glandular secretory apparatus (for example, by inflammatory processes or formations of tumours in the stomach); third, during central or peripheral disturbances in the vago-sympathetic territory. Finally, several of these factors may combine to cause an increase or decrease of HCl.

The conclusion, therefore, is that an anomaly of hydrochloric acid secretion is in itself by no means a certain index of the existence of a stomach disease.

If we further consider that, as has already been pointed out, the limits between normal and abnormal hydrochloric acid secretion are wide, possible conclusions from it can be drawn only with greatest caution, and with painstaking consideration of the other clinical symptoms.

If, then, in the following, we nevertheless discuss the diagnostic significance of the demonstration of hydrochloric acid, it is solely to furnish the practitioner several suggestions.

1. Normal acidity is present (hydrochloric acid value, 0.1 to 0.2 per cent.).

The normal amount of hydrochloric acid speaks, in the first place, against any serious textural disease of the stomach, or against the presence of a stomach disease at all. In obscure dyspeptic disturbances that occur in the wake of all possible internal diseases, such a statement is of practical value.

If, for instance, all other symptoms point directly to a stomach disease, normal hydrochloric acid secretion points in general to nervous (psychogenic) dyspepsia or enteropathy. The demonstration of a normal acidity is of particular value for the differential diagnosis between nervous dyspepsia and chronic glandular gastritis, especially as the subjective symptoms resemble one another enough to cause mistakes. It is to be remembered in this connection that cases of inacidity founded on a nervous basis (achylia gastrica) occur not infrequently.

Furthermore a normal condition of the secretion may be found in atony or hypotony of the musculature of the stomach.

2. Subacidity (hypochlorhydria, hydrochloric acid secretion below 0.1 per cent.).

If subacidity is constantly demonstrated, one may think of a subacute or chronic gastritis; decrease of HCl may, however, occur in ulcus ventriculi or duodeni, in incipient carcinoma, in ectasia and atony of the stomach, in regurgitation of the bile into the stomach, and other conditions.

3. Superacidity (hyperchlorhydria, hydrochloric acid secretion above 0.2 per cent.) is present.

The same may be an important symptom of pyrosis hydrochlorica; or may be the effect of a benign glandular hypertrophy. It may also occur at the beginning of a chronic gastritis (so-called gastritis acida); most commonly, however, it is the symptom of a gastric neurosis. Furthermore, hyperchlorhydria is found in a considerable number of ulcer cases, although this is by no means always the fact. Further, it is frequent in chlorosis. Finally, it is observed in individuals who eat much meat and highly seasoned food.

It is to be remarked, however, that the clinical features of superacidity may be present without an increase of the hydrochloric acid secretion above normal (Verhaegen,[95] L. Schüler[96]).

If undoubted symptoms of carcinoma are present (emaciation, tumour, etc.), Rosenheim[97] considers the presence of free hydrochloric acid at the height of digestion, or hyperchlorhydria, as an indication of the development of this malignant neoplasm from an ulcer, particularly when previous symptoms of an ulcer were present.

4. Inacidity (anacidity, achlorhydria) is present.

Primary inacidity is the most common symptom of the advanced stage of chronic gastritis and of the so-called achylia.

It is further, according to some authors, a not uncommon symptom accompanying gastric neurosis. It is, however, not positive whether a gastritis may not also exist in such cases. Sometimes, however, the determination of the hydrochloric acid deficit, and more so, the digestion and lab-tests (see below, p. 205), will show that no grave stomach disease is present. It is therefore necessary that in cases of achlorhydria, these tests be made.

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[95] Verhaegen, Cellule, T. xv, Fasc. 2; Ref. in Centralblatt für innere Medicin, 1900, S. 106.

[96] L. Schüler, Deutsche medicinische Wochenschrift, 1900, No. 19.

[97] Rosenheim, Zeitschrift für klin. Medicin, 1890, Bd. 17, 135.

The much-mooted question of the absence of HCl in carcinoma ventriculi I will formulate as follows, relying upon my large experience:—

(*a*) In general, the *positive proof* of the existence of free hydrochloric acid speaks *against* carcinoma, but only in association with other symptoms (absence of cachexia, failure of tumour appearance, stagnation, dilatation, etc.).

(*b*) The absence of free hydrochloric acid speaks for carcinoma when at least two other of its classical symptoms (tumour, emaciation) are present, and in all likelihood, when, in company with cachexia, signs of pyloric stenosis and lactic acid formation are present, and when the rest of the clinical course points to the suggestion of a carcinoma.

(*c*) In the differential diagnosis between ulcer and carcinoma, the positive finding of the hydrochloric acid test, the absence of a tumour and of pyloric stenosis indicate the probable presence of an ulcer; the negative finding of the same indicates that ulcers are most likely absent. There are, however, many exceptions to this rule.

5. Alternating acidity (heterochylia, Hemmeter) is present.

This anomaly, observed under the same experimental conditions, points with probability to a gastric neurosis, or a gastritis not too far advanced. Now and then variations of the hydrochloric acid secretions are also seen in carcinoma of the stomach (G. Korn[98]).

In *secondary* dyspepsia (phthisis pulmonalis, cardiac valvular disease, diabetes mellitus, nephritis, liver affections, etc.), investigations of the condition of hydrochloric acid—as is to be expected on the grounds mentioned above—did not yield any useful *diagnostic* hints. In individual cases the same may be of more value as an index for the dietetic treatment.

Organic Acids.

1. Lactic Acid ($C_3H_6O_3$).

Two kinds of lactic acid are found in the stomach: fermentative (æthylidene-) lactic acid (optically inactive), and sarcolactic or paralactic acid (optically inactive).

The former is a product of the fermentation of carbohydrates under the action of bacteria (bacterium lacticum), while the sarcolactic acid is ingested as such or as its salts in muscle-meat, or also in the large glands (liver, spleen, pancreas, lungs, thymus, etc.).

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[98] G. Korn, Archiv für Verdauungskrankheiten, 1902, Bd. 8, Heft 1.

Only the *fermentative lactic acid* possesses a special interest for the diagnosis of stomach diseases.

Tests for Lactic Acid.

1. *Uffelmann's Tests.*[99]—(*a*) One mixes 10 cubic centimetres of a 4 per cent. carbolic acid solution, with 20 cubic centimetres of water, and adds a drop of iron chloride solution, whereupon the mixture takes on an amethyst-blue colour. The mixture must always be freshly prepared, for in a few minutes it assumes a fawn-gray colour. In dilution to 0.1 per cent., lactic acid produces a canary- or lemon-yellow colour. Up to a few years ago I used an iron chloride-carbolic solution made of 3 drops ferric chloride and 3 drops of pure concentrated alcoholic carbolic acid solution, and diluted with water to an amethyst-blue colour (about 200 cubic centimetres). This test is now neglected in favour of that of *b*, which is certainly not inferior.

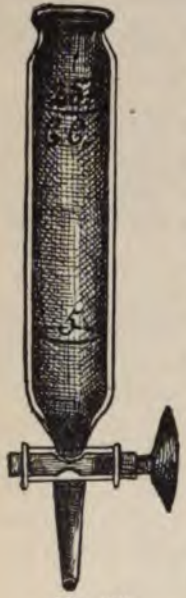

Fig. 26.

(*b*) The same colouring is obtained when lactic acid is added to a highly diluted solution of iron chloride (1 drop liquor ferri to 20 cubc centimetres of water).

(*c*) A few cubic centimetres of filtered gastric contents after addition of some drops of phosphoric acid, are shaken with 50 to 100 cubic centimetres of neutral sulphuric ether, preferably in a separating funnel, and the top layer of ether is evaporated on a hot-water bath. The residue is taken up with some water and tested with the test *b*. In such a case, according to my experience, one proceeds best by mixing the residue with 5 cubic centimetres of distilled water, evaporating the same over a flame to a small remainder, and then adding from a dropper 1 or 2 drops of iron chloride. Even traces of lactic acid are recognised by the canary-yellow colouring which is immediately shown. According to R. Fleischer,[100] the ether needs not be evaporated, but the reagent may be added directly to the ether, which, after agitation, will, in the presence of lactic acid, produce the yellowish colouration of the test-tube. I can recommend this simplified method as very useful.

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[99] Uffelmann, Deutsches Archiv für klin. Medicin, 1880, Bd. 26, S. 431, and Zeitschrift für klin. Medicin, 1884, Bd. 8, S. 393.

[100] Quoted by Penzoldt, Deutsches Archiv für klin. Medicin, 1890, Bd. 51, S. 544.

(*d*) *Modification after Kelling.*[101]—The filtrate of the gastric contents is diluted to ten or twenty times its volume, and the diluted gastric contents treated with one or two drops of a 5 per cent. iron chloride solution. A greenish colour with transmitted light shows the presence of lactic acid, as lactic acid in a dilution of 1 : 10,000 to 15,000 still shows distinctly greenish colour. This method also has proved itself very useful.

(*e*) *Modification after H. Strauss.*[102]—One takes a little shaker-funnel (Fig. 26) that exhibits two marks, one at 5 cubic centimetres and the other at 25 cubic centimetres. The funnel is filled to the mark of 5 with gastric contents, and ether poured over it to the mark 25; it is then shaken, and by turning the stopcock on the funnel the liquid is run off to the mark 5, and the funnel then refilled to the mark 25 with distilled water; 2 drops of iron chloride solution (1 to 9 of water) are now added and vigourously shaken. At about 1 per mille of lactic acid, there occurs an intensely green colour; at a smaller amount, a light green colour. This modification is recommended by some authors as thoroughly answering the purpose.

Sources of Error in Uffelmann's Test.

(*a*) Phosphates and mineral acids in strong concentration, grape-sugar, alcohol, peptone solutions, and other substances give a similar reaction.

(*b*) A high percentage of HCl (2.5 to 3 per mille) hides the lactic acid reaction. This is, however, practically without significance, as a high percentage of HCl excludes the presence of a large amount of lactic acid. If this should, however, exceptionally be the case, one can proceed according to Haas[103] by gradually diluting the sample with distilled water, whereby the disturbing influence of the HCl becomes eliminated.

(*c*) Other acids that appear in the stomach (formic, acetic, butyric) also produce partly a similar reaction as does lactic acid, partly a precipitation.

(*d*) According to Kelling,[104] bicarbonates give, with iron chloride, a straw-yellow to yellowish-brown or brown colour.

(*e*) Sulphocyanides give, with iron, a brown colour. The brownish-red colour which one meets in some gastric contents after the addition of diluted iron solution is, according to the investigations of Kelling, actually to be traced to the presence of sulphocyanide.

(*f*) Furthermore, various foods (meat, egg-products, vegetable substances, milk, various cakes and pastries) may, according to Penzoldt,[105] give Uffelmann's reaction in various degrees.

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[101] Kelling, Zeitschrift für physiol. Chemie, 1893, Bd. 18.

[102] Strauss, Berliner klin. Wochenschrift, 1895, No. 37.

[103] Haas, Münchener medicin. Wochenschrift, 1886, No. 6.

[104] Cr. Kelling, *l.c.*

[105] Penzoldt, Deutsches Archiv für klin. Medicin, 1894, Bd. 53, S. 221.

The consequence is that in case the test is positive, the above-mentioned substances must be excluded; but also if the result of the test is negative, the absence of lactic acid is by no means demonstrated; in all such cases one must substantiate this at least by the establishment of Uffelmann's test with the ether residue.

The lactic acid reaction is produced also by the lactates, but practically this is of but little interest. If necessary, one may demonstrate the presence of the lactates by decomposing them into their component parts after addition of mineral acids.

2. Hoffmann and Vollhardt[106] have employed a method previously offered by Berthelot, for the estimation of lactic acid in the gastric contents, and have reached satisfactory results, though with certain restrictions. This method was used by Richet[107] for the analysis of the gastric contents; its accuracy was questioned by Ewald,[108] on the ground of control investigations. The method depends upon the principle that acids dissolved in water, and agitated with ether, go over with the latter in definite proportion, and that independently from the duration of shaking. If the amount of acid which remains in the water is divided by the amount of acid which goes over into the ether, one gets a definite figure.

This quotient Berthelot called "coefficient de partage."[109] According to Richet, this amounts to 10, Ewald[110] estimated it as 7.8, and Hoffmann and Vollhardt found on the average 10.4. This method gives, however, an accurate value only if, at the shaking, *fermentative lactic acid* alone is to be accounted for as going over into the ether. As, however, in fermentations in the stomach, we have to deal also with other organic fatty acids, the method is impracticable for the above purpose.

3. *Detection of the lactic acid by production of its salts.*—The simplest is the production of the zinc salt. The proceeding is as follows: First, the albuminous bodies are coagulated by boiling (eventually with addition of dilute sulphuric or nitric acid) and filtered; the filtrate, after addition of a small amount of barium carbonate, is brought on a water bath to the consistency of a thin syrup. The syrup is treated with several portions of absolute alcohol, allowed to remain for some length of time, and then filtered. The filtrate is again evaporated to a small volume, acidulated with a few drops of phosphoric acid, and taken up with a copious portion of neutral alcohol-free ether. After standing for some time, the

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[106] F. A. Hoffmann and Vollhardt, Archiv für experimentelle Pathologie and Pharmakologie, 1891, Bd. 28, Heft 5 and 6, S. 423.

[107] Richet, Du suc gastrique chez l'homme et les animaux, Paris, 1878.

[108] Ewald, Virchow's Archiv, 1882, Bd. 80.

[109] Berthelot, Annales de chimie et de physique, Bd. 26, 4 Serie, p. 396.

[110] Ewald, *l.c.*

clear ether stratum is taken off, the ether is removed, the acid residue, after the addition of a freshly precipitated zinc carbonate, is boiled with water, then filtered, and concentrated to a small volume. On cooling, the zinc lactate crystallises in beautiful rhombic crystals, single or united in groups (Fig. 27).

The zinc lactate is hardly soluble in cold water, rather easily soluble in hot water, almost insoluble in alcohol. To aid the identification, a definite amount is weighed, and is weighed again at 120° C. until the constant weight is reached. The loss of weight, corresponding to the water of crystallisation of the fermentative zinc lactate, must amount to 18.18 per cent. (The calcium salt of fermentative lactic acid is soluble in 9.5 parts of water, and on heating to 100° C. it loses 29.2 per cent. water of crystallisation, while the calcium salt of the sarcolactic acid is soluble in 12.4 parts of water and loses 26.21 per cent. of water of crystallisation).

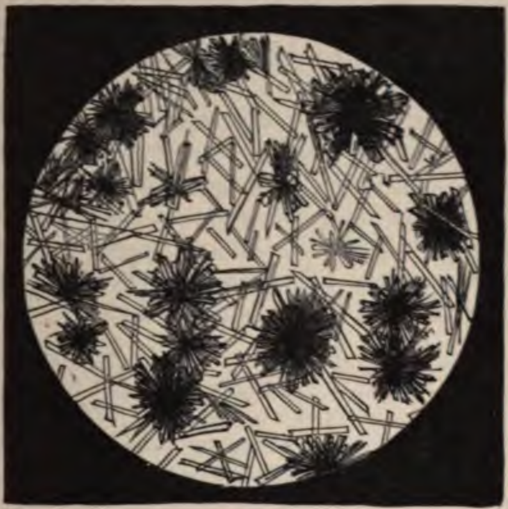

Fig. 27.

For the detection of sarcolactic acid one proceeds in the same manner. For the identification of the zinc salt one must heat to 100 to 120° C.; the sarcolactate of zinc loses 12.9 per cent. of water of crystallisation.

Test for Lactic Acid According to Boas.[111]

Principle of the Method.—When solutions of sarcolactic as well as fermentative lactic acid are treated with strongly oxidising substances and heated, they are decomposed into acetaldehyde and formic acid according to the following formula:—

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[111] Boas, Deutsche medicinische Wochenschrift, 1893, No. 34.

$$\underset{\text{Lactic acid}}{CH_3 - CH(OH) - COOH} = \underset{\text{Acetaldehyde}}{CH_3 - CHO} + \underset{\text{Formic acid}}{CHOOH}.$$

If under considerable heating a very strenuous oxidation occurs, the named products are not the end-products, but the process goes further, so that the aldehyde is converted into acetic acid, the formic acid into carbonic acid and water, according to the following formula:—

$$\underset{\text{Aldehyde}}{CH_3 - CHO} + \underset{\text{Formic acid}}{CHOOH} + 2O = \underset{\text{Acetic acid}}{CH_3 - COOH} + CO_2 + H_2O.$$

By a careful and gradual heating, however, one can control the process of oxidation in such a way that a conversion of aldehyde into acetic acid can be avoided.

The proof of lactic acid is accordingly carried on simply by the detection of one or the other of the products (acetaldehyde or formic acid). The simplest and easiest is the detection of the acetaldehyde.

As qualitative reaction of aldehyde, an alkaline iodine solution is used, which forms with aldehyde, similarly as with alcohol and acetone, iodoform (Lieben's reaction). This method also serves, as will be explained later, for the quantitative estimation of lactic acid.

Proceeding for the Qualitative Test of Lactic Acid.—Ten to 20 cubic centimetres of the stomach-contents are evaporated in a porcelain dish on the water-bath to the consistency of a syrup. No further addition is required when free acid is absent, but when it is present, baryta carbonate in excess is added. Then the syrup is treated with a few drops of phosphoric acid, and, after the carbonic acid has been driven off by boiling, is allowed to cool, and repeatedly extracted with small portions (2 or 3 times each 50 cubic centimetres) of absolutely alcohol-free ether (distilled with sodium).*

After a half-hour's treatment, the clear stratum of ether is poured off, the ether evaporated, the residue is taken up, with 45 cubic centimetres of water, into a retort, thoroughly shaken and filtered, and the filtrate is mixed with 5 cubic centimetres of concentrated sulphuric acid (specific gravity, 1.84), and as much manganese as will go on the point of a knife is added. The retort is corked with a perforated, well-fitting stopper, through the perforation of which goes a glass tube, bent in an obtuse angle. Its longer limb dips into a small cylinder which contains 5 to 10 cubic centimetres of an alkaline iodine solution (that is, equal parts of a decinormal iodine solution and normal potassium solution). The fluid to be tested is heated over a small flame, and when lactic acid is present, the aldehyde goes over into the iodine at the very beginning of boiling, and very soon the so-called iodoform reaction occurs (clouding and odour of iodoform, iodoform crystals).

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* The extraction with ether is for the purpose of eliminating the carbohydrates, which themselves, when oxidised, partly supply aldehyde.

Quantitative Estimation of Lactic Acid.

1. According to my experience, one can approximately estimate the amount of lactic acid in the contents of a stomach by comparing the yellow colouring obtained by the use of Uffelmann's test with a solution of known (as weak as possible) amount of lactic acid. By gradual dilution one may reach the same tint, and thus draw an approximate conclusion as to the amount of lactic acid in the gastric filtrate. If, for instance, one gets a distinct greenish-yellow colouration after addition of one or two drops of a 5 per cent. solution of iron chloride to stomach-contents which had been diluted ten or twenty times, there is, according to Kelling, approximately 1 per mille or 2 per mille lactic acid present.

2. More exact is the following procedure, which I have tested for a long time. Mix the filtrate with a few drops of dilute sulphuric acid, and, after heating over a flame, whereby the albuminous bodies are coagulated, filter and evaporate over a water bath to the consistency of syrup; refill to the original volume, and again evaporate to a small volume. Hereby the volatile acids have been removed, and the residue contains only lactic acid. This is now extracted with a large amount of ether (200 cubic centimetres of ether to 10 cubic centimetres); the ether is evaporated, and the residue is taken up by water, and titrated with phenolphthalein and decinormal potassium solution. Each cubic centimetre of the decinormal lye used represents 0.009 gramme lactic acid. By this procedure, a part of the lactic acid which escapes with heating or evaporation, is lost.

3. The direct weighing, after the manufacturing of the salt concerned, is said to give very exact values (according to the statement of Palm,[112] the preparation of the lead salt is the best adapted). The same is dried and glowed, whereby lead oxide is left. As the compound contains 78.5 per cent. of lead oxide and 21.5 per cent. of lactic acid it is needful only to multiply the weight of the residue, after glowing, with $\frac{21.5}{78.5} = 0.274$. According to the investigations of de Jong,[113] the method is, however, inexact and very tedious.

4. The method for qualitative test for lactic acid described by me is also adapted for an exact quantitative estimation of lactic acid. The procedure is as described, the only difference being that the ether residue, which was taken up by water (45 cubic centimetres) and treated with manganese and sulphuric acid (5 cubic centimetres), is distilled under careful cooling. Very appropriately the retort is furnished with a doubly-perforated stopper, through one perforation of which there is a bent glass tube leading to the cooler, and in the other there is a second tube, likewise bent and furnished

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[112] Palm, Zeitschrift für analyt. Chemie, Bd. 26, S. 33.

[113] de Jong, Archiv für Verdauungskrankheiten, 1896, Bd. 2, S. 69.

with a short rubber tube and clamp; the latter glass tube is to drive out any aldehyde in the retort or cooler by means of a current of air.

The distillation is continued until about four-fifths of the liquid under test have gone over.

The lower end of the cooler is furnished with a bent glass tube that dips into a retort containing about 20 cubic centimetres of water, and which preferably stands in a dish filled with ice.

The lactic acid is now simply estimated by means of titration.

For carrying out the titration the following solutions are necessary:—

1. Decinormal iodine solution.

2. Decinormal sodium arsenite solution (or, instead of it, decinormal thiosulphate solution).

3. Hydrochloric acid of specific gravity 1.018.

4. Potassium lye (about 56 grammes potassium hydroxide to a litre of water).

5. A thin, freshly prepared starch solution.

The alkaline iodine solution is added to the distillate—usually 10 to 20 cubic centimetres of decinormal iodine solution dissolved in 20 cubic centimetres of potassium lye of the above concentration, is sufficient—and the fluid, well shaken and carefully covered, is left alone for several minutes. In order to set free iodine out of the potassium iodide and subiodide which did not enter in action, the sample is mixed with 20 cubic centimetres of the normal hydrochloric acid solution, an excess of sodium bicarbonate is added, and titrated with the sodium arsenite from a burette till entire decolourisation ensues.* The possible excess of sodium arsenite is titrated back under the addition of a freshly prepared starch solution by means of decinormal iodine solution; at the first appearance of a permanent blue colour the end reaction is reached. The number of cubic centimetres of decinormal iodine solution minus the number of cubic centimetres of decinormal arsenious acid solution shows the amount of iodine which was necessary for the formation of iodoform, and indirectly the amount of lactic acid.

The calculation of the lactic acid depends on the fact discovered by me, that 1 cubic centimetre of decinormal iodine corresponds to 0.003388[*] gramme of lactic acid,[**] so that in order to estimate the lactic acid one needs only to multiply the resulting number of cubic centimetres of decinormal iodine with the said figure.

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* The estimation of the amount of iodine through arsenious acid depends, as is well known, on the oxidising power that iodine exerts upon oxidisable substances: the arsenious acid is converted into arsenic acid, while the iodine is converted into hydroiodic acid, so that the originally reddish-brown colour of the iodine solution is decolourised. From the amount of arsenious acid which must be used, we can draw a conclusion as to the amount of iodine contained in solution. As we are working with decinormal solutions, every cubic centimetre of $^1/_{10}$ $AsO_3H_3 = 1$ cubic centimetre of $^1/_{10}$ I. An excess of sodium bicarbonate is added because the reaction between arsenious acid and iodine follows smoothly and quickly only in an alkaline solution. As, however, caustic alkalies and monocarbonates of alkalies themselves act upon the iodine, only bicarbonates of alkalies can be employed for the alkalinisation.

Example:—

Original.................10 c.cm. of deci-normal iodine.[***]
Used for titration...6.5 c.cm. of decinormal arsenious acid.
For titrating back..........0.2 c.cm. of decinormal iodine.

consequently

Iodine, total..................................10.2 c.cm.
Arsenic, total................................ 6.5 c.cm.

According to this, 3.7 c.cm. of decinormal iodine is needed for the formation of iodoform, from which it follows that the lactic acid amounts to 3.7 × 0.003388 = 0.0125 for ten cubic centimetres, or 1.25 per mille.

Precautions in Tests for Lactic Acid.

If one wishes to test for the presence of lactic acid in the gastric contents with a view to diagnostic purposes, one must first of all *distinguish between lactic acid introduced into the stomach and that formed in the stomach.* Lactic acid is ingested with meat as sarcolactic acid; besides, fermentative lactic acid is frequently ingested with the diet, for example, with milk, especially sour milk, buttermilk, sour kraut, grape-juice, lemon-juice, various sorts of fruits, sugars, etc. It is worthy of remark that all our baked products, especially rye (schwarz) bread, but also, as I could prove,[114] light (wheat) bread, zwieback, crackers, all contain a more or less large amount of lactic acid. The Ewald-Boas test-breakfast, otherwise so well adapted, is on this account less useful for scientific purposes. I have found meal-soup (best made from Knorr's oat-meal) an absolutely lactic-acid-free diet article. If one wishes to examine quantitatively the formation of lactic acid in the stomach, oat-meal soup (½ litre) to which only salt has been added, is recommended as best. If stagnation is found in the morning, it is best, according to my experience, to thoroughly wash out the stomach late at night, and then to let the patient take the said quantity of meal-soup. Any meal-soup residue found the next morning before breakfast is then adapted for the test for lactic acid. For the purposes of daily practice, however, the test-breakfast may be satisfactory; but, as Bönninger[115] has recently proved, the Riegel test-meal is not adapted, on account of its containing sarcolactic acid.

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[*] According to de Jong (Archiv für Verdauungskrankheiten, 1896, Bd. 2, S. 71) 1 cubic centimetre of decinormal iodine solution corresponds to 0.0030 gramme of lactic acid.

[**] 14.4 cubic centimetres of decinormal iodine solution represent 0.00239 gramme acetaldehyde, from which follows that 1 gramme acetaldehyde corresponds to 602.5 cubic centimetres of decinormal iodine solution. Further, 90 grammes of lactic acid correspond to 44 grammes of aldehyde. From this follows the proportion, $44 : 90 = 0.0239 : x$; $x = 0.0488$; that is, 0.0239 gramme aldehyde corresponds to 0.0488 of lactic acid; consequently 14.4 cubic centimetres of decinormal iodine solution correspond to 0.0488 of lactic acid, or 1 cubic centimetre of decinormal iodine solution corresponds to 0.003388 gramme of lactic acid.

[***] For 10 cubic centimetres of gastric contents to be examined.

[114] Boas, *l.c.*

Practical Value of the Various Methods of Lactic Acid Estimation.

One may be satisfied with Uffelmann's test, as soon as this yields an undoubted result, that is, when the colour turns an *intense green.* Especially worthy of recommendation for controlling the result are the above-mentioned modifications of Fleischer, Kelling, and Strauss. All tints other than green are in general worthless, for they speak neither for nor against the presence of lactic acid. This holds good not merely for the test-breakfast, but also for the meal-soup recommended by me, in the employment of which I have repeatedly observed a slightly yellow colour, although lactic acid could not be substantiated by the aldehyde test. Also the extraction by shaking up with ether cannot, according to my experience, alter this.

For scientific purposes the aldehyde test is a reliable and relatively simple method.

Very much more circumstantial is the formation of a lactate and determination of the water of crystallisation, besides being applicable only with a high amount of lactic acid. For absolutely exact estimation it surpasses, however, the aldehyde test, in which a slight loss of lactic acid is hardly avoidable.

Diagnostic Significance of the Lactic Acid Test.

To judge of the diagnostic significance of lactic acid, the question first of all to be settled is under what circumstances and conditions lactic acid presents itself in the stomach-contents. It has already been pointed out that, as Ewald[116] has shown, and Martius and Lüttke[117] and myself[118] have substantiated, lactic acid does not occur at all after the ingestion of carbohydrates in healthy individuals. In like manner, I have been able to prove that also in benign affections of the stomach—gastritis, ectasia, atony, neuroses, etc.—lactic acid is not found at all, or only in traces. As furthermore, lactic acid fermentation does not occur in the presence of free hydrochloric acid, according to general consent, the investigation for lactic acid has neither practical nor theoretical value in these cases. Practically always, however, lactic acid production is associated with stagnation and absence of hydrochloric acid.

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[115] Bönninger, Deutsche medicinische Wochenschrift, 1902, No. 41.

Now, in so far as both of these factors are most frequently found in gastric carcinoma, the lactic acid test is an extraordinarily valuable diagnostic aid for the recognition of this disease. Its significance is limited only by the facts that, (1) lactic acid is by no means constantly present in gastric carcinoma, for many cases run their course to the end without stagnation; (2) that secretion of hydrochloric acid is also met with in gastric cancer with stagnation, much more rarely without stagnation; (3) that there are undoubtedly other pathologic conditions of the stomach, in which abnormal lactic acid fermentation may occur (hypertrophic stenosis of the pylorus, corrosions of the stomach, atrophy of the gastric mucous membrane, etc.).

Nevertheless, the finding of lactic acid offers valuable, and sometimes decisive, evidence as to the existence of a carcinoma of the stomach even in those cases in which no tumour is palpable. Now and then one can establish the diagnosis of a gastric cancer at a very early stage on the ground of the chemical findings, and thus also induce an early operation. In spite of absence of a tumour, laparotomy may, however, reveal an advanced carcinoma accompanied by metastases. (Compare also Part II, p. 571.)

2. Volatile Fatty Acids.

Volatile fatty acids—apart from their ingestion with the diet—may occur as products of anomalous carbohydrate fermentation; particularly butyric acid and acetic acid.

The test for the volatile fatty acids can, for practical purposes and preliminary showing, be carried out in this manner. About 10 cubic centimetres of the gastric contents are heated in a test tube, at the end of which a small strip of blue litmus paper has been put. On the evaporation of the volatile fatty acids the litmus paper is reddened (Leo[119]).

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[116] Ewald, Virchow's Archiv, 1882, Bd. 90.

[117] Martius and Lüttke, Die Magensaüre des Menschen, Stuttgart, 1892.

[118] Boas, Münchener medicinische Wochenschrift, 1892, No. 43.

(a) *Butyric Acid ($C_4H_8O_2$).*

1. *By the odour.*—At the presence of only a slight admixture of butyric acid there is given off the well-known acrid odour. This may, however, be obscured, when other volatile acids are present.

2. A small quantity (10 cubic centimetres) is taken up with 50 cubic centimetres of ether, well shaken up, and the ether is evaporated. The residue is taken up with a trace of water, and on the addition of a small amount of calcium chloride (best in substance), shows a separation of butyric acid in the form of small oil-globules, which at the same time give the specific odour of butyric acid. By saturation with baryta water one gets, on evaporation, the production of baryta butyrate in rhomboid platelets or granular warts refracting light like fat.

3. *Method of Cahn and v. Mering.*—The butyric acid is demonstrated in the distillate, as just described.

4. *Method of v. Jaksch.*[120]—The gastric contents are distilled with phosphoric acid; the distillate is carefully neutralised with solution of carbonate of sodium and evaporated to dryness, extracted with hot alcohol, filtered, and the filtrate evaporated to the smallest volume, and tested for butyric acid in the following manner: on the addition of mineral acids the offensively smelling butyric acid is split off from the butyrates; solution of iron chloride does not give a red colour, and silver nitrate brings about a crystalline precipitate that is not soluble in water.

5. *Method of Hoppe-Seyler.*—The gastric contents are freed from albuminous bodies by means of alcohol, and filtered; the filtrate, made alkaline with sodium carbonate, is evaporated to a small volume, and the residue, after the addition of diluted sulphuric acid, is distilled. The distillate is made alkaline, brought to a small volume, and, after addition of an excess of acid, again distilled. From the second distillate thus concentrated the butyric acid, on addition of $CaCl_2$, can be separated as oil. Mixture with propionic acid is recognised by fractional distillation. The latter boils at a temperature as low as 140°, while butyric acid boils between 150° and 163° C. From the purest possible butyric acid thus obtained the silver salt may be formed. Silver butyrate ($C_4H_7AgO_2$) requires 55.38 per cent. Ag.

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[119] Leo, Diagnostik der Krankheiten der Bauchorgane, Berlin, 1895, P. 316.

[120] v. Jaksch, Klinische Diagnostik innerer Krankheiten, 5 Aufl., 1901.

(b) Acetic Acid ($C_2H_4O_2$).

1. Even in minimal quantities the presence of acetic acid manifests itself through the well-known pungent odour.

2. A small amount of gastric contents is extracted with acid-free ether, and evaporated; the residue, taken up with a few drops of water, is exactly neutralised with a solution of carbonate of sodium. At the presence of acetic acid sodium acetate is formed from the free acid, which gives, with diluted iron chloride, a deep bloody-red colour. Furthermore, silver nitrate produces a precipitate which is soluble in hot water.

3. A test-sample is extracted with ether, and the residue is likewise neutralised with a solution of carbonate of sodium. At the presence of acetic acid there occurs, on heating with some sulphuric acid and alcohol, the characteristic odour of acetic ether.

Diagnostic Significance of the Volatile Fatty Acids.

The diagnostic significance of the volatile acids is, as far as known, the same as that of lactic acid. According to the investigations of Leney and Vaughan,[121] they are to be found in especially large amounts, in cases of hypochlorhydria, but were found within normal limits in some cases of reduced motility as long as the secretion of hydrochloric acid remained normal

Examination of Enzymes.

1. Pepsinogen and Pepsine.

The pepsinogen, the specific product of the chief cells of the glands in the stomach, is characterised by the fact that through the action of acids, with especial rapidity through HCl, it is converted into active pepsine, whereby it becomes enabled to convert proteids and gelatines into their soluble modifications.

In this statement, the practical test for pepsine and pepsinogen is given. If the gastric contents contain free acids and digest proteids, the proof of the presence of pepsine is given. If the contents contain no free acid, the digestive power of the same, when treated with sufficient HCl (see below), speaks for the presence of pepsinogen; the lack of digestive power, for the absence of the same.

Neutral gastric contents alone, however, without artificial addition of hydrochloric acid, is by no means under all conditions incapable of digestion, but, on the contrary, according to my experiences, digestive power of such gastric filtrates is not frequently found. In such cases we have to deal with an admixture of pancreatic juice and bile. Such a state of affairs shows a sufficiency of pancreatic digestion, a fact which, under certain circumstances, may be of diagnostic and prognostic significance.

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[121] Leney and Vaughan, British Medical Journal, 1899, Bd. 27, P. 1271; Virchow, Hirsch. Jahresb., 1899, Bd. 1, S. 147.

Directions for the Pepsinogen and Pepsine Tests.

1. *When free HCl is present:—*

(*a*) *Qualitative test.*—Ten cubic centimetres of gastric contents are put in a test-tube and a little disk of egg-albumen or a dried fibrine flake (preparation of Dr. Grübler, in Leipzig) or lamellæ of serum albumen (albumen e sanguine),* all if possible of a definite weight, is added and the test-tube is placed in an incubator which either is provided with a thermoregulator** or at least can be maintained at a constant temperature of between 37° and 40° C. Solution of the albumen after a short time speaks for the presence of pepsine.

Of practical moment is the fact that egg-albumen takes a considerably longer time for solution than fibrine and serum-albumen do.

According to Jaworski, a disk of egg-albumen weighing 5 to 6 centigrammes in 25 cubic centimetres stomach filtrate is digested in three hours at a temperature of 40° C. The same quantity of fibrine under the same conditions is dissolved in one-half the time, while 5 to 6 centigrammes of serum-albumen are dissolved to a slight remainder in one hour.

(*b*) *Quantitative Test.*—We do not possess as yet a pepsine-estimation practically useful and at the same time accurate; we must be satisfied with approximate estimations. For this purpose we may simply follow v. Leube's directions, that we take two samples of filtrate, and add to one of them a small quantity of powdered pepsine and the same amount of albumen. If the sample to which pepsine has been added dissolves the albumen more rapidly, pepsine is deficient; if it shows the same peptic power, pepsine is present in sufficient quantity.

In my experience the following procedure is as valuable: I use as a standard the gastric contents of a healthy person, which I preserve and the digestive powers of which I know. I compare with this the digestive powers of the sample to be tested. If, for instance, the former digests a definite amount of albumen in one hour, and the latter in two, three, or more hours, the digestive power of the test gastric filtrate is one-half, one-third, one-fourth of the normal standard filtrate. This will suffice for practical purposes.

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* For sale at Merck Co., Darmstadt [and New York].

** The thermoregulator is a desideratum for scientific purposes; for practical purposes, a simple sheet-metal chamber, with a double floor, is entirely satisfactory.

One may also use the colourimetric procedure offered by Grützner for physiological purposes, in such a manner that one mixes carmine fibrine with the filtrates in question and that the red colouring of the mixture is compared with samples of known amounts of pepsine.

2. *When hydrochloric acid is absent.*

In these cases *pepsinogen* alone, or at least predominantly, is found in the stomach-contents. Pepsinogen is tested for by acidulating 10 cubic centimetres of the gastric filtrate with officinal hydrochloric acid (one or two drops) and proceeding as with pepsine. For quantitative estimation of the amount of pepsinogen, I proceed similarly as with lab-zymogen (see below). I dilute the gastric contents with distilled water in the proportions of 1 : 5, 1 : 10, 1 : 20, etc. The higher the dilution that does not interfere with the digestive powers, the richer is the amount of pepsinogen.

Exactly the same method has been recently used by Johannessen[122] without his arriving at stringent results.

If the contents acidified with hydrochloric acid does not digest at all, *pepsinogen* also is absent.

Hammerschlag[123] has recently offered a method for the quantitative estimation of pepsine, or, more correctly, the peptonising power of the gastric contents, which is to be used for the purpose of practice. The method depends upon the fact that two samples of 10 cubic centimetres each are taken from a 1 per cent. albumen solution* which also contains 3 to 4 per mille *free* hydrochloric acid; to the one sample are added 5 cubic centimetres of water and to the other the same number of cubic centimetres of the stomach-contents the peptic power of which is to be tested. After an *hour's standing* in the thermostat, and after *twenty-four hours' standing* in Esbach's albuminimeter, the amount of albumen of each sample is determined in the well-known manner. The control sample mixed with water gives the original amount of albumen; the difference between the two represents the amount of digested albumen. If, for example, the control sample mixed with water contains 6 per mille of albumen, that with gastric juice 3 per mille, 50 per cent. of the albumen has been digested. This method of Hammerschlag has been criticised by Oppler,[124] Roth,[125] Troller,[126] and especially by Schorlemmer,[127] but we cannot here go into a discussion of the errors pointed out. It is certain, however, that the method is insufficient for scientific investigation, and for practical purposes it is to be used only with great precaution.

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* To prepare the solution, it is best to proceed by carefully rubbing 3 grammes of the commercial dry egg-albumen with a little lukewarm water in the mortar, then filling with water to 150 to 180 grammes; filter the whole, and add so much officinal hydrochloric acid that the mixture by titration with decinormal lye, with the use of dimethylamidobenzol as indicator, contains 3 to 4 per mille of free HCl.

[122] Johannessen, Zeitschrift für klin. Medicin, 1890, Bd. 17, Heft 3 and 4.

[123] Hammerschlag, Internationale klin. Rundschau, 1894, No. 39.

Instead of the freshly prepared albumen solution, Troller used 1 per cent. protogen solution in decinormal HCl, Strauss uses filtered puro-juice* with decinormal HCl as standard solution.

Method of Oppler.—Oppler[128] has worked out a very much more exact method, which, however, is unfortunately too complicated for practical purposes. The principle is to determine the amount of digested albumen according to the well-known Kjeldahl's method.

In detail, the process is as follows: Early in the morning, before breakfast, the patient takes 60 grammes of light bread and 400 cubic centimetres of water, after food-remnants have been thoroughly removed from the stomach. After one hour the contents are expressed, and *the stomach washed with small portions of water* until the water comes away clear. They are now filtered, and then water added to one or two litres, and then brought to an acidity of 77 with diluted hydrochloric acid. First 50 cubic centimetres of this liquid are now examined for nitrogen estimation. Another 50 cubic centimetres of the same liquid are mixed with 20 cubic centimetres of about 2 per cent. albumen solution, and digested in two closed retorts for three hours in the thermostat, then quickly neutralised with proper solution of NaOH, boiled, acidified with acetic acid, and, after an addition of 5 cubic centimetres of saturated NaCl solution, again boiled. After cooling, the coagulated albumen and liquid are washed with the smallest possible amount of distilled water, so that the total amount reaches 150 cubic centimetres, which is then filtered. Fifty cubic centimetres of the filtrate are now used for the N estimation according to Kjeldahl. One may use as standard liquid a 1 per mille pepsine solution, which, according to Oppler's investigations, digests 35 to 38 per cent. of albumen in three hours. If, according to my suggestion, such a solution is designated as normal pepsine solution, it is evident that one can compare the amount of pepsine, after the result of the peptic action has been obtained, with this normal pepsine solution. If, for instance, the gastric contents under examination (undiluted) digests 12 per cent. of albumen in three hours, the peptic power corresponds to one-third normal pepsine.**

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* [Puro is a German meat preparation, similar to Valentine meat-juice.]

** For further details, see the thorough and careful contribution of Oppler, Archiv für Verdauungskrankheiten, 1902, Bd. 8, Heft 3-5.

[124] Oppler, Archiv für Verdauungskrankheiten, 1896, Bd. 2, S. 40.

[125] Roth, Zeitschrift für klin. Medicin, 1900, Bd. 39, S. 1 u. 2.

[126] Troller, Archiv für Verdauungskrankheiten, 1899, Bd. 5, S. 751.

[127] Schorlemmer, *ibid.*, 1902, Bd. 8, S. 299; Berliner klinische Wochenschrift, 1902, No. 51.

[128] Oppler, *l.c.*

Method of Mett.—The already-described method of Mett has been highly recommended as a useful clinical method by Linossier,[129] Roth,[130] L. Kuttner,[131] and Schorlemmer.[132] But, on the other hand, it does not lack opponents. Troller,[133] for instance, denies every significance of Mett's method, on account of irregular digestion, and A. Schiff[134] also is very non-committal concerning its practical utility. In recent years, Nirenstein and Schiff[135] have pointed out a very noteworthy source of errors in Mett's method, namely, that carbohydrates and common salt considerably hinder the digestion in Mett's glass tubules, and that in an uncontrollable manner. These authors, however, found that this hindering influence is entirely eliminated through diluting the gastric contents to sixteen times its volume.

Nirenstein and Schiff, therefore, recommend for clinical purposes the following modification: one cubic centimetre of the filtrate of gastric contents is diluted with 15 cubic centimetres of $^1/_{20}$ normal hydrochloric acid to sixteen times its volume. Mett's tubules are placed in a sample of a thoroughly shaken mixture. After twenty-four hours, the digested columns are read off, which have to serve directly as the comparative standard for the amount of pepsine in the stomach-contents.

A numerical estimation of the relative amounts of pepsine is also made possible through the digestive values found after the method of Nirenstein and Schiff. This is obtained by squaring the read-off values of the pepsine amount and by multiplying with 16 the relative amount of the native juice. For instance, gastric contents X (diluted 16 times) digests 1.78 millimetres; therefore the relative amount of the diluted juice $1.78^2 = 3.17$, and the relative amount of the original juice $16 \times 3.17 = 50.72$.

2. Lab-Zymogen and Lab-Ferment.

[Rennin-zymogen and Rennin.]

Qualitative Test for Rennin.—Five to ten cubic centimetres of stomach-filtrate are accurately neutralised with decinormal lye, and then, with the addition of the same quantity of neutral or amphoteric reacting raw or, better, boiled milk, placed in an incubator. If within ten to fifteen minutes curdling sets in, and if on further standing a coagulum (cheese) is formed, the coagulation is to be attributed to the action of rennin. To make sure of this, the reaction of the mixture is tested before and after; it must not have changed during the act of coagulation. Much simpler and better adapted for practical purposes is the following method, of which Leo[136] makes use: after addition of 3 to 5 drops of the gastric contents to 5 to 10 cubic centimetres of milk, the same is placed in an incubator; if coagulation follows within ten to fifteen minutes, lab-ferment is present. The presence of a slight amount of acid does not come into account, though it is questionable whether this test suffices when only a slight amount of enzyme is present.

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[129] Linossier, Journal de physiol. et de pathol. génér., 1899, Bd. 1, Heft 2.

[130] Roth, Zeitschrift für klin. Medicin, 1900, Bd. 39, Heft 1 u. 2.

[131] L. Kuttner, *ibid.*, 1902, Bd. 45.

[132] Schorlemmer, Archiv für Verdauungskrankheiten, 1902, Bd. 8, Heft 3-5.

[133] Troller, *ibid.*, 1899, Bd. 5.

[134] Schiff, *ibid.*, 1900, Bd. 6.

[135] Nirenstein and Schiff, *ibid.*, 1902, Bd. 8, Heft 6.

For the purpose of testing for *lab-zymogen* 10 cubic centimetres of weakly alkaline stomach-filtrate are treated with 2 or 3 cubic centimetres of a 1 per cent. calcium chloride solution, or neutralised to a *slight* alkalescence with lime-water; and then, after the addition of 1 cubic centimetre of milk, the mixture is placed in a thermostat or in a dish with water of 37° C. In case of the presence of lab-zymogen there occurs within a few minutes the formation of a thick coagulum of caseine.

Quantitative Test for Rennin.—When the gastric contents are gradually diluted, the final limits of a distinct action of the lab are reached. In detail, the method of procedure is as follows: first a test for lab-enzyme is made in the manner described above. If the test reacts positive, the neutralised stomach-contents is diluted to one-tenth, one-twentieth, one-thirtieth, etc. When the limit has been reached, the remainder is made slightly alkaline with potassium lye or soda, and the examination for lab-zymogen is made in the same manner.

For lab-enzyme, the dilution limit is under normal conditions, about $^1/_{30}$ to $^1/_{40}$; for the zymogen, $^1/_{100}$ to $^1/_{150}$.*

Meunier[137] measures the amount of lab-ferment by the time that elapses until coagulation sets in. According to my experience, however, the time of coagulation-action depends, besides on other factors, essentially on the degree of acidity in the stomach-contents.

Diagnostic Significance of the Test for Enzymes in the Gastric Contents.

While the amount of free hydrochloric acid in the gastric contents is subject to the greatest variations, due to the above-mentioned conditions, (p. 185) and therefore can be used for diagnostic purposes only with great caution, the ferments of the stomach or their preformations show, as a rule, more constant values. Anomalies of the circulation of the blood or of the innervation do not seem to influence them in a marked degree, so that *a pronounced diminution of their specific biologic action is directly indicative of a disturbance of the function of the glandular apparatus itself.*

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* In this connection, of course, the error due to the dilution of the gastric contents during neutralisation is to be taken into account in the calculation.

[136] Leo, Berliner klinische Wochenschrift, 1888, No. 49.

[137] Meunier, Journ. de chim. et de pharm., Bd. 12, S. 457-465; Ref. in Maly's Jahresbericht, 1900, S. 414.

The necessity of possessing a criterion independent of varying acidity is evident, especially in the frequent cases of inacidity. We do not possess, up to the present, the possibility of forming an idea from the absence of acidity alone whether in an individual case we have to deal with a catarrh *ex retentione,* or with an incipient and curable gastritis, or an advanced incurable catarrh, or, finally, with a chronic disturbance of innervation. The consequence is that any safe basis for the prognosis and therapy is absent.

Through the examination of the enzymes, however, we may judge in individual cases whether a transitory or a permanent impairment of the glandular apparatus is present. It is known, for instance, that in anomalies of menstruation, in nervous dyspepsia, in congestive conditions, in acute and in early stages of chronic gastritis, the hydrochloric acid secretion is temporarily inhibited. The test for enzymes gives us in these cases a prompt answer to the question whether we have to deal with a temporary suppression of hydrochloric acid or with an advanced gastritis. To be sure, Oppler,[138] in his valuable work, has demonstrated that a certain parallelism exists between hydrochloric acid secretion, pepsine, and the presence of lab, but Oppler's investigations were concerned only with cases with constant disturbances of the hydrochloric acid secretion. Just for the transitory variations of the same we possess, in the test for enzyme, a good control for the type and the degree of the disturbance. For examination of the condition of the pepsine a qualitative test suffices, with a normal stomach-contents as control (see above), or the method of Mett, or also that of Hammerschlag, is satisfactory. It is important to know that according to the investigations of Oppler, even in atrophy of the gastric mucous membrane pepsine is still secreted to a slight degree. The observation made by Hammerschlag,[139] that in carcinoma of the stomach, pepsine disappears early, is accurate only partially, as, according to Roth, Schiff, and Schorlemmer, good peptic juices have been found in cases of carcinoma. Only in those cases associated with loss of hydrochloric acid, diminution of the albumen-digesting power was the rule.

The quantitative estimation of rennin leads to especially useful results. Through experiments which I carried out,[140] partly in association with Dr. Trzebenski and partly alone, it has been established:—

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[138] Oppler, Archiv für Verdauungskrankheiten, 1896, Bd. 2.

[139] Hammerschlag, Archiv für Verdauungskrankheiten, 1896, B. 2.

1. That lab-enzyme may be present even if HCl be absent; but only in exceedingly small quantities, namely, at a limit of a dilution of 1 : 10 to 1 : 20.

2. That in spite of achlorhydria the zymogen may be preserved to its entire extent, namely, the limit of a dilution of 1 : 100 to 1 : 150. *The proof of the presence of lab-zymogen, particularly if this is the result on repeated examinations, permits in all likelihood the conclusion that an organic affection of the stomach is not present,* but that we have to deal with either a neurosis or passive congestion, due to primary affections of various kinds. If the investigations further prove that hydrochloric acid is absent temporarily, but reappears at other times, it likely indicates a gastric neurosis.

3. The zymogen may be diminished about half. The cause of this is most commonly to be found in a catarrhal condition that is not yet advanced to an extreme degree. The more the amount of zymogen approaches the normal, the greater is the possibility of a restitution under the influence of proper therapeutic measures.

4. The formation of lab-zymogen is diminished to a great extent (dilution of 1 : 10 to 1 : 25 yields negative results), or is entirely absent. In cases of this kind there exists a grave, usually incurable catarrh; it does not matter whether the catarrh is idiopathic or secondary (carcinoma, amyloid, etc.).

5. In conditions 1, 2, and 3, it is worth a trial to stimulate the HCl secretion by appropriate measures, and that with prospect of results. In condition 4, such a thing seems to be absolutely impossible.

The *test for rennin* has undoubted advantages over the *peptic test,* which are of practical value. The same are to be found in the following facts: (1) That the result may be obtained quickly; (2) that the test may be performed with the least possible amount of gastric filtrate; (3) that in an alkaline reaction of the gastric filtrate (saliva, mucus, enteric juice) it is possible to facilitate the decision as to whether gastric secretion is actually present in the mixture; (4) that no special preparation is required, and may be carried out at any time with materials found in the household.

Bile, mucus, and saliva have a disturbing influence upon the test for rennin only when the mixture reacts alkaline; in neutral or acid reaction an influence worthy of note is not to be observed. In the former case, however, at least, the test for lab-zymogen can be performed in the manner named above.

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[140] Boas, Deutsche medicinische Wochenschrift, 1892, No. 17.

Glässner[141] has recently called attention to an interesting, as well as practically important antagonism between the lab-ferment and pepsine. Relying upon the experience that the pyloric section of the stomach secretes only pepsine, while the fundus secretes lab-ferment and pepsine, Glässner was able to state that in carcinoma of the fundus pepsine and lab-ferment are diminished or are absent, while in carcinoma of the pylorus the amount of lab-ferment present is normal, but that of pepsine is reduced.

Concerning the condition of the fat-splitting ferment in gastric affections, only a few contributions have been made. Volhard[142] *found pronounced diminution of the ferment in cases of achylia. Higher degrees of hyperchlorhydria inhibit also the secretion of the fat-splitting ferment. As according to Volhard's investigations, the fat-splitting ferment is produced only in the fundus but not in the pyloric section, an extension of Glässner's investigations in this direction would be of great interest.*

Examination of Proteids in the Gastric Contents.

The proteids are peptonised in various intermediary stages under the influence of the gastric juice, especially of the combination of pepsine and hydrochloric acid. Hydrochloric acid may also be replaced by lactic, butyric, and other fatty acids. Besides this, micro-organisms are found in the saliva, and consequently in the stomach also, which by a long action upon the proteids and under favourable conditions of propagation may split off peptonoids (Miller).

Between the proteids and the final peptones, there are, as has already been mentioned (p. 30), several intermediary products, among which syntonin and propeptone, or hemialbumose can be distinguished by characteristic reactions.

As under certain circumstances we may have to consider the question whether the proteids have been digested perfectly or imperfectly, completely or only partly, the course of investigation may be briefly described as follows:—

1. Tests for *syntonin* or acid albumen are made by neutralising the filtrate as accurately as possible. When syntonin is contained in large amounts, there occurs on neutralisation a more or less pronounced cloudiness, or even a precipitate (neutralisation precipitate), which dissolves on addition of an alkali or an excess of acid.

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[141] Glässner, Berliner klin. Wochenschrift, 1902, No. 29 and 32.

[142] Volhard, Verhandlungen des 19 Kongresses für innere Medicin, 1901, p. 302; Zeitschrift für klin. Medicin, 1901, Bd. 43. In this contribution there will also be found the somewhat circumstantial technique which demands the appliances of a laboratory; we cannot discuss the same at this time.

2. *Propeptone* or hemialbumose is characteristic in that respect, that concentrated acetic acid and saturated sodium chloride solution in excess produces a pronounced cloudiness, which disappears on heating and reappears on cooling. Likewise an excess of nitric acid produces pronounced cloudiness that dissolves on heating with the formation of a yellow colour (xanthoprotein reaction) and reappears on cooling. Before instituting the reaction for propeptone it is necessary to eliminate any albumen precipitated by boiling. Syntonin and propeptone give a cloudiness with acetic acid and potassium ferrocyanide, and picric acid precipitates all proteids in the cold with the exception of true peptones; but on heating the precipitate of hemialbumose is dissolved, while the other proteins remain undissolved.

Propeptone, peptone, as well as all albumoses in general, exhibit with an excess of potassium lye and copper sulphate solution (0.1 : 100), the so-called *biuret reaction,* namely, a beautiful pink colour that turns a very dark red on the further addition of copper solution. Syntonin, as well as all the albuminous bodies lying between it and the hemialbumose, exhibit the biuret reaction either weakly or not at all; that is, the colour shows immediately a characteristic violet tint which quickly turns bluish on further addition. According to this, the biuret reaction has a certain practical significance, since it indicates the presence of a peptic action. After some practice it may also be possible to recognise whether there has been a sufficient or insufficient action of the gastric juice on proteid substances.

The reactions of the remaining albumen derivatives formed during gastric digestion have been described above (p. 30).

In order to obtain the peptones pure out of solutions, we proceed by first eliminating globulin and syntonin by boiling and a possible addition of acetic acid; and by treating the filtrate with concentrated neutral ammonium sulphate solution in excess, all albuminous bodies, with the exception of peptones are precipitated. Some length of time is needed (at least twenty-four hours) before all the albumen bodies are gradually precipitated. A thorough separation of the albuminous bodies from the peptone takes place in a short space of time according to Devoto's method:[143] the fluid containing albumen is mixed in a beaker up to 100 cubic centimetres with 80 grammes of crystalline chemically pure ammonium sulphate, and the salt is completely dissolved by heating (in a water bath, stirring and crushing the crystals with a glass rod), which takes from ten to fifteen minutes. Then the beaker is exposed to boiling water for thirty to forty minutes, whereupon coagulation is completed. If we let the beaker linger in the steam for a longer time, up to two hours, the coagulum becomes denser, and the filtering and washing can be performed much quicker.

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[143] L. Devoto, Zeitschrift für physiologische Chemie, 1891, Bd. 15, Heft 5, S. 465.

The filtrate now gives no reaction with acetic acid and potassium ferrocyanide, nor with neutral salts, picric acid, trichloracetic acid, etc.; it is therefore free from albumose, but still exhibits the biuret reaction.

Diagnostic Conclusions Drawn from the Condition of the Protein Bodies in the Gastric Contents.

As the conversion of the proteid bodies, as already mentioned, depends essentially upon the presence of pepsine and hydrochloric acid, the demonstration of albumoses indicates in general only the fact that HCl, and as a matter of course also pepsine, has been secreted.

This, therefore, we might consider only as a complicated test for HCl and pepsine. The value of the albumose reaction is still further impaired by the above-mentioned fact that organic acids also may produce the same, and that, furthermore, a digestion of desquamated cell-substances likewise leads to the formation of peptone, and that, finally, albumoses or similar substances may be due to bacterial decompositions.*

Thus it happens that there is hardly a gastric filtrate that does not give a more or less pronounced biuret reaction. *Accordingly, the reaction by itself is not sufficient proof for the occurrence of a normal proteid digestion,* although the sharp and distinct positive result of a pink or purple colour can be used by the experienced observer as an approximate measure of the presence or amount of peptones. If, however, according to the above method, true peptones are found to be present, even if only in a small amount, the action of HCl and pepsine may be considered as proven.

A large amount of syntonin and the lower albumoses speaks for an anomalous course of proteid digestion, and the same is also the case when the biuret reaction entirely disappears after the removal of the albumoses. This fact signifies either a defective secretion of HCl or a simultaneous diminution of enzymes.

Examination of Carbohydrate Digestion in the Gastric Contents.

The saccharification of the starches, as has already been mentioned (p. 19), passes through three intermediary stages that vary characteristically in their attitude towards iodine-potassium iodide (Lugol solution): amidulin, erythrodextrine and achroödextrine; the first gives a blue colour with iodine-potassium iodide, the erythrodextrine a violet to mahogany-brown, while achroödextrine remains uncoloured.

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* It should not be forgotten that, owing to the manner of preparation (boiling of milk, eggs, meat, etc.), a part of the albumen has already been changed to albumoses.

As a rule, one finds in gastric contents containing much HCl after ingestion of carbohydrates, even towards the end of digestion, an abundance of erythrodextrine, while in the same length of time in a contents free of HCl, we get almost exclusively an achroödextrine reaction.

There is an exception to this rule when the salivary glands are diseased, so that the pytaline has suffered a partial or total loss of activity, as is occasionally the case (p. 129).

The end product of the conversion of starch into sugar is maltose ($C_{12}H_{22}O_{11} + H_2O$) or isomaltose, together with small amounts of dextrose, demonstrable by the known tests (most practically, Fehling's or Nylander's).

Cane-sugar is quickly converted by muriatic gastric contents into invert-sugar. v. Leube[144] is of the opinion that such is the case only in pathologic conditions. This is, according to my (unpublished) investigations, by no means the case. The normal gastric contents also may, in proportion to the acid secretion, which I consider as the sole acting agent, convert cane-sugar into dextrose and lævulose, only that it leaves the stomach very quickly by resorption, while in disturbances of the stomach it is demonstrable for a longer time. In certain cases, the acid-free contents may convert also cane-sugar. This depends, it seems to me, on bacterial changes which, as we know according to Miller's investigations, even under physiologic conditions, occur already in the oral cavity.

Noteworthy investigations concerning the changes of other kinds of sugar in the stomach have not been reported.

Diagnostic Significance of the Examination of Carbohydrates.

The digestion of carbohydrates is of significance only in so far as it supplements the quantitative examination of gastric acidity. In most cases the reaction of the carbohydrates toward iodine solution is indirectly almost as certain a chemical reagent for the presence or absence of acidity as the qualitative tests for it; for in hyperchlorhydria, according to experience, the digestion of amylum is considerably impaired (with iodine, a pronounced blue colouring), while it is manifested to a considerable extent in achlorhydria (wine-yellow colouring with iodine). Only the previously mentioned cases of deficiency of ptyaline (p. 129) suggest caution as to conclusions, and cause the HCl tests to be necessary. Under certain circumstances both excess of acidity and deficiency of ptyaline may work together.

The test for sugar is hardly valuable for diagnostic purposes, particularly when food containing sugar is ingested. When sugar-free carbohydrates have been ingested, a moderate amount of sugar would indicate an impairment of the action of the salivary diastase, which may be caused, for example, by abnormal HCl secretion or by deficiency of ptyaline. If cane-sugar is inverted in HCl-free contents, it would, according to my investigations, probably be due to bacterial action.

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[144] v. Leube, Virchow's Archiv, 1882, Bd. 88, S. 222.

Examination of the Motor Function of the Stomach.

We can determine the motor function of the stomach either by introducing food of which we know, by experience, in what length of time—apart from physiologic variations—the same leaves the stomach; or we may proceed by introducing into the stomach definite food-substances or medicines that are not dissolved or not absorbed in the stomach, but which are dissolved in the small intestine, and which may be determined after a given time, qualitatively or quantitatively, either directly (in the stomach-contents) or indirectly (in the urine). After the various methods will have been detailed in the following, their value will be critically reviewed.

(a) By Means of v. Leube's Test-Meal.[145]

For investigation the following test-meal is necessary (corresponding with the Riegel test-meal): A plate of soup, a beefsteak, a roll, and a glass of water; or, more accurately, according to Riegel, 400 cubic centimetres of beef-broth, 200 grammes of beefsteak, 50 grammes of bread, and 200 cubic centimetres of water. The patient must not eat anything during the following seven hours. At the end of this time the stomach is washed out, according to v. Leube, in such a manner that the funnel is filled twice with about half a litre of water. If no food-remnants appear or only a very few, the motor function of the stomach is to be regarded as sufficient.

(b) By Means of the Test-Breakfast.

The motor function of the stomach may be determined, to be sure not exactly, but sufficiently for practical purposes, by means of the test-breakfast, that is, if the precaution is observed that it be ingested under the supervision of the physician. Under normal conditions the test-breakfast leaves the stomach in two hours at the latest; so that if at the end of this time, it is found that large quantities of fluid or food-remnants are present, the motor function of the stomach may be regarded as insufficient.

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[145] v. Leube, Spezielle Diagnose der inneren Krankheiten, 1889, P. 232.

(c) *By Means of Test-Supper.*[146]

At eight o'clock in the evening the patient takes a meal consisting of 400 grammes of tea with milk and sugar, two pieces of white bread with butter, and cold meat ad libitum. Until the expression or washing-out of the stomach, which takes place the next morning, the patient is not to take food or drink. If under these circumstances remnants of the gastric contents are found in the fasting stomach, a high-grade motor insufficiency (insufficiency of the second degree) is present.

If no remnants are present, a slight motor insufficiency may nevertheless be existing, which may be best demonstrated by means of v. Leube's test-meal, or by means of estimation of the residue according to Mathieu-Rémond (p. 151).

(d) *Method of Ewald and Sievers.*[147]

This method depends upon the principle that salol, a phenol ester of salicylic acid, is not dissolved in acid media, but dissolves in alkaline media (therefore, in the small intestine), whereby it is decomposed into phenol and salicylic acid. The latter is excreted in the urine as salicyluric acid, and may be easily recognised by iron chloride solution (violet colour). The investigation is best carried out by the ingestion of 1 gramme of salol (preferably given in a wafer, for in powder form a certain part may be decomposed even in the oral cavity). Under normal conditions the salicylic acid reaction begins to appear in the urine in forty to sixty minutes, or at the latest in seventy-five minutes, while a later occurrence would point to a disturbance of the motility of the stomach. Marked objections have been raised against the usefulness of the salol test by various observers (Brunner,[148] Huber,[149] Decker,[150] Wotitzky,[151] Reale and Grande,[152] Stein[153]). According to the experiments of Reale and Grande and H. Stein, the method has lost in value, for they proved that salol may also decompose in the stomach, especially under the influence of the gastric mucus. Furthermore, it seems noteworthy that J. Pal[154] proved that the decomposition of salol depends also on the condition of the intestine. According to this author, the test is reliable only in one case, namely, when the decomposition product is absolutely absent in the urine, a factor which would speak in all likelihood for an absolute occlusion of the pylorus.

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[146] Boas, Deutsche medicinische Wochenschrift, 1894, No. 28.

[147] Ewald and Sievers, Therapeutische Monatshefte, August, 1887.

[148] Brunner, Deutsche medicinische Wochenschrift, 1889, No. 7.

[149] Huber, Münchener medicinische Wochenschrift, 1889, No. 7.

[150] Decker, Berliner klinische Wochenschrift, 1889, No. 45.

[151] Wotitzky, Prager medicinische Wochenschrift, 1891, No. 31.

[152] Reale and Grande, Rivist. clinic, October, 1891.

[153] Stein, Wiener medicinische Wochenschrift, 1892, No. 43.

[154] J. Pal, Wiener klinische Wochenschrift, 1889, No. 48.

Modification of the Salol Test according to A. Huber.[155]

On account of the variations that occur thereby, Huber does not determine the first appearance of salicylic elimination in the urine, but the *length of time of its continuance.* In healthy persons this lasts twenty-six to twenty-seven hours, while in patients with gastric insufficiency there may be an excess of three to twelve hours, or even more. According to the directions of Huber, the patient takes 1 gramme of salol in a wafer after dinner, and the urine is examined the following day, after about twenty-seven hours. If at this time the iron chloride still gives a violet colour, the urine is further examined every three hours. According to Huber, the length of the duration of the reaction seems to be directly proportional to the degree of motor insufficiency.

Silberstein[156] investigated Huber's modification of the salol test and likewise arrived at satisfactory results. On the other hand, Wotitzky[157] showed that in patients with healthy stomachs the salicylic reaction may last longer than twenty-seven hours, and that, on the contrary, in sick patients, and indeed in those with considerably impaired motility (stenosis of the pylorus after ulcer, carcinoma of the pylorus), this time may not be exceeded in some cases.

(e) Method of Fleischer.[158]

One decigramme of iodoform in a little gelatine capsule is taken on an empty stomach directly before the beginning of the chosen meal. As the iodoform is not soluble in acid media, but is soluble in alkaline, we have in the appearance of the iodine reaction in the urine or saliva an expression of the rapidity with which the substance is removed from the stomach. According to the investigations of Maurer and Kypke-Burchardi,[159] the iodine reaction is found in healthy persons in the majority of cases after fifty-five to sixty-five minutes, but may be delayed to 105 minutes. In patients affected with severe disturbances of motility, the reaction seldom appears before the lapse of three to four or still more hours.

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[155] A. Huber, Korrespondenzblatt für Schweizer Aerzte, 1890.

[156] L. Silberstein, Deutsche medicinische Wochenschrift, 1891, No. 9.

[157] Wotitzky, *l.c.*

[158] Fleischer, Krankheiten der Speiseröhre, des Magens und Darms, Wiesbaden, 1896, P. 791.

[159] Quoted by Fleischer.

(f) Method of Winkler and Stein.[160]

It was first established by Winternitz that iodipin is not split into its component parts by the gastric contents after even the lapse of a very long time, while bile and pancreatic secretion act upon it very rapidly. Upon this principle Winkler and Stein founded their method of testing the motility of the stomach.

The person to be examined takes one teaspoonful of iodipin one-fourth to one-half hour after a test-breakfast. The iodine reaction is tested in the saliva fifteen minutes later, and if negative the examination is carried on until positive results occur. For the test of the iodine, Winkler and Stein, after the recommendation of Bourget, use starch-paste paper which has been saturated (in the dark) with ammonium persulphate (5 per cent.). The slightest traces of iodine call forth a blue to black colour.

By means of the iodipin method, Winkler and Stein found that, under normal relations, the iodine reaction sets in one-fourth hour at the earliest and three-fourths hour at the latest. Delay of the iodine reaction occurred in gastroptosis, dilatation of the stomach, and carcinoma of the stomach.

The method has been substantiated by many authors (Sternberg, Peterson, Schupfer, Lubicelli, Heichelheim, Werner). From this it is to be seen that the method is to be considered as useful, especially when, for any reason, an examination of the gastric contents is contraindicated.

(g) Method of Klemperer.[161]

This method depends on the fact that fat is not absorbed at all from the stomach, and is altered only slightly. After the ingestion of a certain quantity of oil, the amount of oil obtained by aspiration after a given time yields a definite expression of the motor function of the stomach. In detail the procedure is this: 100 grammes of olive oil are introduced through the stomach-tube into the empty or washed-out stomach. One should really introduce 105 grammes, as about 5 grammes are lost in the introduction. The gastric contents are aspirated after about two hours, whereby the greater portion of the oil still remaining in the stomach is recovered, and the remainder is obtained by repeated washing with water. The combined amounts of liquid are united, and the oil separated from the water in the separating funnel; the remaining oil, which is mixed with water and mucus, is taken up with ether, the ether evaporated, and the remaining oil is weighed.

By this method Klemperer found that in many healthy persons the normal quantity of oil which had escaped was 70 to 80 grammes, while when the motor function was reduced, this amount was considerably smaller. Klemperer does not wish to recommend this test for daily practice, but thinks it should serve rather to establish the condition of the motor function in a typical manner in various gastric affections. Thus the founder of the method has drawn strict limits for its usefulness.

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[160] Winkler and Stein, Centralblatt für innere Medicin, 1899, No. 33.

[161] Klemperer, Deutsche medicinische Wochenschrift, 1888, No. 47.

(h) Method of A. Mathieu.[162]

This method depends in principle upon Klemperer's oil method, with the additional basis of Mathieu-Rémond's estimation of the remaining stomach-fluid. In detail the proceeding is as follows: the patient, fasting, takes 60 grammes of white bread, and in addition the following emulsion: Ol. amygd. dulc., 10.0 cubic centimetres; gum arabic, 5.0 to 10.0 cubic centimetres; syr. simpl., 20.0 cubic centimetres; weak tea enough to make a total of 250 cubic centimetres. Expression an hour later. Then 200 cubic centimetres of distilled water are poured through the funnel, recovered, and again used, in order to get an intimate mixture of the water with the remaining gastric fluid. From this fluid, the amount of fluid remaining in the stomach after expression is determined according to Mathieu-Rémond's formula (p. 150). In the undiluted sample we further determine, by extraction with ether and weighing of the fat after evaporation of the ether, the amount of fat contained in the same; and as we now know the combined amount of fluid we determine the total amount of oil.

(i) Method of E. Goldschmidt.[163]

This method aims to find the amount of fluid that escapes from the stomach in a given time. It depends, in principle, upon the procedure suggested by H. Strauss for estimating the remaining amount of gastric contents; that is, the amount of contents not recovered by expression, by means of determination, according to an already-stated formula (p. 154) of the specific gravity of the undiluted gastric juice, and that remaining in the stomach, which is to be diluted in a definite manner. According to Goldschmidt, the method is faulty because the aræometer scales necessary for the determination of the specific gravity are far from being accurate.

Goldschmidt, therefore, offered the following relatively simple procedure for estimating the amount of the liquid gastric contents:—

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[162] A. Mathieu, Archiv für Verdauungskrankheiten, 1895, Bd. 1, P. 345.

[163] E. Goldschmidt, Münchener medicin. Wochenschrift, 1897, No. 13.

After the ingestion of a certain amount of liquid, it is expressed after a definite time. After expression, the remaining portion of the contents is diluted with a definite amount of water (50 cubic centimetres of distilled water); after mixing the fluid in the stomach, expression is repeated. Both fluids, the undiluted (*a*) and the diluted (*b*), are filtered, and the specific gravity of *b* taken with an aræometer. Now as many cubic centimetres of distilled water as were previously used (50 cubic centimetres) are put into a glass cylinder, the filtrate of the undiluted gastric juice is put into a burette, and allowed to drop by cubic centimetres into the cylinder with the 50 cubic centimetres of water until the aræometer indicates exactly the same specific gravity as the diluted gastric juice shows. The number of cubic centimetres used, which we now read from the burette, represents the exact number of cubic centimetres that remained behind in the stomach after the first expression.

(k) Method of Schüle.[164]

Schüle's procedure depends on the estimation of the dry residue after the ingestion of a definite meal. In detail, Schüle proceeds as follows: one hour after a test-breakfast or three hours after a test-dinner, the contents are expressed, and the stomach is thoroughly washed. After chemical and microscopic examination of the gastric contents, the combined contents (including wash-water) are freed of the greater part of the liquid by decantation, and then filtered through a previously weighed filter. The contents are left standing for several days, and then weighed when air-dried. Thus we learn how much of undigested contents remained in the stomach after such a test-meal. The results attained vary, as was to be expected, in normal as well as in pathologic cases. For example, the residue of a test-breakfast varies under normal conditions between 1.0 and 5.5 grammes. It is evident that this method cannot be of practical value for judging the motor function, because the dissolved substances, which likewise belong to the residue, are hereby left out of account. Besides, the method is too circumstantial for practical purposes.

(l) Method of Sahli.[165]

Sahli's procedure depends on the following train of thought: if we succeed in finding a substance which, when added to the test-meal, is not absorbed in the stomach and which is very easily estimated quantitatively, it may be determined, after the recovery, from the amount of substance remaining, how much remained in the stomach and how great that part of the recovered contents is, which actually consists of secretion. For this purpose, Sahli gives 300 cubic centimetres of a flour-soup which contains butter-fat in uniform dissemination.* After three-fourths to one hour it is expressed, and the amount and the residue are determined according to Mathieu and Rémond. From the dilution of the soup, calculated by the fat-percentage of the recovered portion (by Gerber's lactobutyrometer), the amount of secreted juice that had been present in the stomach at the time of expression may be determined. By calculation, the acidity of the pure secretion is then obtained from the total acidity of the portion drawn off. This method is, as may be seen, rather complicated, but this would not be an insuperable obstacle. Unfortunately, as the congruent experiments of v. Koziczkowsky[166] andBöniger[167] have shown, the determination of fat gives unequal results, due to the sedimentation of the recovered flour-soup. It is, therefore, not astonishing that quite dissimilar values were arrived at in the same individuals on different days. The method fails because of unequal distribution of the fat in the soup.

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[164] Schüle, Fortschritte der Medicin, 1901, Bd. 19, P. 445.

[165] Sahli, Berliner klinische Wochenschrift, 1902, No. 16 and 17; Seiler, Deutsches Archiv für klin. Medicin, 1901-1902, Bd. 71 and 72.

Practical Value of the Individual Methods.

None of the above methods serves as an ideal of an exact estimation of the function of the stomach, although undoubtedly they may be of value for clinical judgment in individual cases. Decidedly the most suitable is v. Leube's method, particularly because it gives an expression of the physiologic activity of the stomach. Although the time of expulsion accepted by v. Leube may now and again vary somewhat even under normal conditions, in general it may be regarded as the mean time for physiologic gastric motor function. The test-supper suggested by myself may also be utilised as a valuable measure for determining quickly and quite positively severe disturbances of motility. Both methods supplement each other, in so far as v. Leube's test-meal is decisive as to the normal or abnormal condition of the motility, while the test-supper gives also information as to the degree of motor disturbance.

As to the other methods, there is no doubt that motor disturbances may be determined by any of them. We must not, however, attach too much importance to their value. This refers especially to the salol test, and probably also to the iodoform and iodopin methods. Klemperer's oil method is undoubtedly accurate physiologically, but in practice it meets with insuperable difficulties; and the procedure of Mathieu is also too complex for practical purposes. Goldschmidt's procedure is also not without objections, because the expressed fluid and that remaining in the stomach do not represent only that which was introduced, but have also been augmented by secreted gastric juice, or by bile, mucus, transudate, etc., so that we are dealing with uncalculable values. It is self-evident, although the results on many cases may be approximate, that an absolutely reliable expression for the motility is not to be obtained by this method. On the other hand, the same seems to me very useful as a method for estimation of the residue. The just-mentioned method of Sahli is, apart from its sources of error, too complex for employment outside of hospitals.

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*[Flour is put into a pan, in which butter has been melted, and the whole is stirred constantly till the flour becomes brownish-yellow; the necessary amount of water is then added and the whole is allowed to come to quick boiling.]

[166] v. Koziczkowsky, Deutsche medicinische Wochenschrift, 1902, No. 26.

[167] Böniger, Münchener medicinische Wochenschrift, 1902, No. 43.

Causes of Motor Weakness.

Insufficiency of the motor function may be due to various causes: (1) weakness of the expelling force; (2) obstacles to expulsion.

1. *Weakness of the expelling powers* (myasthenia, paresis) may occur:—

(*a*) As a congenital condition (rare cases);

(*b*) As the result of certain constitutional diseases (anæmia, chlorosis, phthisis pulmonalis, leukæmia, diabetes mellitus or insipidus, chronic malaria, amyloid degenerations, syphilis, etc.), or as a sequel of acute infectious diseases (cholera, puerperal fever), and in my experience very frequently after *typhoid fever.* It may also occur in association with chronic gastric catarrh.

(*c*) Myasthenia may occur as an independent affection due to irregular mode of life.

(*d*) Myasthenia is frequently combined with enteroptosis.

(*e*) As result of specific diseases of the musculature of the stomach (due to corrosion or infiltration of a cancerous growth into the musculature). This is especially common in the flattened growth of carcinoma of the smaller curvature or of the anterior wall of the stomach, in which, although physical examination does not show ectasia, the delay of the motor function is so great that food-remnants may be found in the fasting stomach in the morning. Fatty and colloid changes (R. Maier) of the muscle fibres may also influence the mechanism to a greater or lesser extent.

(*f*) Nervous affections not rarely lead to paresis of the musculature, as, for example, neurasthenia, bulimia, polyphagia, local irritation of the nerve-endings, psychoses, etc.

2. *Obstructions to the expulsion of chyme* occur under the following circumstances:—

(*a*) *In the gastric wall itself.*—Here in the first place we have to mention ulcus ventriculi, with its well-known seat of predilection at the pylorus; then carcinoma of the pylorus. In rare cases benign tumours, polypi, etc., may obstruct the outlet. Further, here belong also the likewise rare congenital narrowness of the pylorus, and the hypertrophic thickening of the musculature of the same. Also to be mentioned are gall-stones, which may lodge in the pylorus or the duodenum or their neighborhood.

(*b*) *Causes for stenosing of the pylorus, apart from those lying in the wall of the stomach,* are so many that they cannot all be mentioned. We mention only the most important: tumours near the pylorus, adhesions of the pylorus with the neighbouring organs (gall-bladder, liver, small intestine, pancreas, transverse colon, etc.) or ligamentous constricting bands from the same causes, floating kidney, herniæ, volvulus (Fr. Müller), etc.

Examination of Gastric Absorption.

Method of Penzoldt and Faber.[168]

Two decigrammes of chemically pure (namely, iodic acid-free) potassium iodide, best in a gelatine capsule from which all traces of the iodide have been wiped off, are administered to the individual under investigation a short time before a meal. In intervals of two or three minutes the saliva or urine is tested for the presence of iodine by means of starch-paste paper and fuming nitric acid. In healthy persons, there appears after six and one-half to eleven minutes a violet, and after seven and one-half to fifteen minutes a bluish reaction. If the drug is given after the meal the absorption-time is considerably delayed (to forty-five minutes for the blue reaction. Penzoldt and Faber, Zweifel, Sticker, Quetsch, and others).

In pathological disturbances of the stomach, according to the investigations of Penzoldt, Faber, Wolff, Quetsch, and Zweifel, the elimination of iodine is considerably delayed. According to Zweifel,[169] to whom credit is due for the most exact investigations, there is a tendency towards delay of absorption in nearly all gastric affections, greatest in dilatation and in cancer of the stomach, slight in chronic catarrh of the stomach, and only little in ulcer.

According to Zweifel, delay to 120 minutes was found in ectasia, to 82 minutes in gastric carcinoma, and in chronic catarrh to 21 minutes, while in ulcus ventriculi the absorption time was partly normal, partly delayed.

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[168] Penzoldt and Faber, Berliner klinische Wochenschrift, 1882, No. 21; and Faber, Diss. Erlangen, 1882.

[169] Zweifel, Deutsches Archiv für klinische Medicin, 1886, Bd. 39, P. 349. (There, also, other literature.)

According to Zweifel, when the absorption time (of 0.2 gramme) of potassium iodide exceeds twenty minutes, gastric dilatation or carcinoma of the pylorus, or both, is to be thought of, provided that an extensive fresh destruction of the gastric mucous membrane can be ruled out; for in the latter case also considerable retardation of the absorption-time may be found. The absorption-time, however, is not, under all circumstances, to be depended upon as to differential diagnosis between gastric cancer and ulcer.

Häberlin[170] also made detailed investigations as to the absorption-time of potassium iodide in carcinoma. He found normal absorption-time in four cases among thirty patients, retardation of the same in 87 per cent. As reason for the same, he found, on autopsy, in the first group a relatively intact mucous membrane, while in the other group he considered the delay due to a diffuse growth of the carcinoma over a wide area of the gastric mucosa, and partly to the consecutive dilatation.

According to this, the iodide of potassium test is neither of differential diagnostic moment for the distinction between ulcer and carcinoma, nor is it of decisive significance for the latter alone.

If we concede that the diagnostic value of the iodide of potassium test is thus shattered, it is still more so after the experiment instituted by v. Mering[171] on dogs with duodenal fistula, whereby he proved that generally iodine salts are not at all absorbed from the gastric mucous membrane. Otto,[172] however, who worked in Penzoldt's laboratory, found that iodides are very well absorbed from the stomachs of guinea-pigs and rabbits. His experiments on dogs agree with those of v. Mering.

Direct proof of the *incapability of absorption* of potassium iodide by the human stomach must still be given, before we can apply the animal experiments to human pathology.

The whole question of absorption, however, is deprived of its diagnostic significance because, on the strength of experiments made on human beings by means of yolk-sugar mixtures, v. Mering found the significant fact *that the absorption shows no difference between normal and pathological conditions.*

Abnormal Constituents of the Gastric Contents.

Abnormal substances which may, under certain circumstances, be of significance for the diagnosis, are not rarely mixed with the gastric contents. First of all, there are among them the following: mucus and saliva, bile and enteric juice, and, finally, blood and pus.

Mucus and Saliva.—As a rule, one can easily distinguish between saliva which has been swallowed from the true gastric mucus by its being mixed together with foam, and by its floating on the gastric contents. Usually it floats on the surface of the gastric contents or in the funnel (Ad. Schmidt[173]). The gastric mucus, however, is intimately mixed with the food-remnants. As Schmidt states, in its isolated and unmixed state it is found only at the end of the lavage. The masses of mucus appear fibrous and frayed, and are found coming up to the top in the washing-funnel; they do not, however, float on the surface, but settle to the bottom on standing. In doubtful cases, the origin of mucus may be established by the microscope; sputum-cells and myelin-drops characterise the bronchial mucus, squamous epithelium characterises that of the mouth, throat, and œsophagus.

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[170] Häberlin, Deutsches Archiv für klinische Medicin, 1889, Bd. 45, P. 347.

[171] v. Mering, Klinisches Jahrbuch, 1899, Bd. 7, P. 3.

[172] Otto, Archiv für Verdauungskrankheiten, 1902, Bd. 8, P. 4 and 5.

One finds a slight amount of mucus even under normal conditions, and this becomes pathologic only when it is intimately and uniformly mixed with the gastric contents. The highest degree of admixture with mucus, as Schmidt has correctly observed, is seen in the absence of the secretion of hydrochloric acid (atrophy of the gastric mucus membrane, carcinoma). On the contrary, little or no mucus is found in neuroses, atonies, and benign stenoses of the pylorus. There is only one exception to this, namely, the so-called gastritis acida, in which, in spite of the distinct presence of the free hydrochloric acid, considerable amounts of mucus may be seen macroscopically (pouring off from one vessel to another). The *chemical* investigation of the *filtrate* of gastric juice (mucin test) is, as Schmidt has shown, not to be considered; it may succeed, perhaps, in subacid or inacid gastric contents, but even then we are left in doubt as to whether we have to do with gastric mucus or with mucus from other organs.

Bile and Enteric Juice.—A slight amount of admixture of bile and enteric juice is found not rarely in the juice of the fasting stomach, as the closure of the pylorus is easily broken through when the stomach is empty. Constant presence of bile and enteric juice speaks for stenosis in the descending portion of the duodenum. The presence of even a minimal amount of bile is shown by the well-known yellowish-green colour of the gastric contents. In case of doubt it is necessary to test for bile by demonstrating either bile-pigments or biliary acids or cholesterin.

The presence of pancreatic secretion may be detected by its specific ferment action. It may be recognised by its tryptic, diastatic, and fat-splitting action.

The *tryptic* action is demonstrated by treating the gastric filtrate, if it reacts acid, with 1 per cent. soda solution until it becomes distinctly alkaline, and then by adding a flake of fibrine. If trypsin is present, the fibrine dissolves, gradually forming peptone after passing through the stages of several intermediary products. At the same time tyrosin and leucin are formed, and a substance not yet clearly defined, but which produces a violet colour with bromine or chlorine water in acid solution.

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[173] Ad. Schmidt, Deutsches Archiv für klinische Medicin, 1896, Bd. 57, P. 65.

To prove the presence of the *diastatic ferment*, the process, analogous to that in the case of the saliva, is to bring starch-paste in contact with filtrate which has been rendered weakly alkaline with Na_2CO_3. After it has been left for a short time in the thermostat, maltose and (only a little) grape-sugar are formed, after passing through the previously-mentioned intermediary stages. In like manner glycogen, too, is changed to sugar under the influence of the pancreatic diastase.

The *fat-splitting* action of the pancreatic juice may be demonstrated in a simple manner, as follows: a few drops of the weakly alkaline filtrate are placed in a watch-crystal, and three or four drops of neutral olive-oil, as well as one or two drops of alcoholic rosolic acid, are added. The mixture now becomes weakly violet to pink. The watch-crystal is now covered with a second crystal and made air-tight with a clamp and put into the incubator. After thirty to sixty minutes the pink colour becomes fainter and fainter, and finally disappears. A fat-splitting may also be brought about through the possible presence of bacteria, but only to a smaller extent, and after a much longer time. Or we may proceed according to Heidenhain,[174] as follows: litmus granules are triturated with milk, which is then filtered through glass fibres. A sample of this blue-coloured milk is then treated with the liquid to be tested. After digesting for some time in a water-bath at 40° C, it becomes red, while a sample prepared in like manner, excepting that it has been boiled beforehand, remains blue.

These simple methods suffice for practical purposes, but to one who wishes to study thoroughly the fat-splitting action of the secreted juice, the procedure of Hoppe-Seyler,[175] adapted also for quantitative estimation, is recommended.

Blood and Pus.—A small amount of fresh blood, or blood not quite recognisable macroscopically, may be determined:—

1. *By Means of the Microscope.*—Red blood-corpuscles may be readily recognised in the acid-free contents, but only sporadically or not at all in blood no longer fresh or entirely decomposed. The demonstration of blood-corpuscles is fully sufficient for the diagnosis.

2. *By Heller's Blood-Test.*—A quantity of gastric filtrate is mixed with the same quantity of normal urine, is made distinctly alkaline, and boiled. The precipitated phosphates assume a garnet-red colour and in thin layers appear dichroitic. On account of admixtures (coffee, cocoa, red-wine, bile, etc.), the contents may have a colour of their own, so that the red colour of the precipitate is not distinctly recognised. It is well, therefore, to collect the precipitate on a small filter, and to dissolve the same in acetic acid. Hereby the fluid assumes a red colour, which when exposed to the air gradually disappears.

This test may be used with advantage on decomposed blood, but it must be remembered that rhubarb, senna, and santonin show a similar colouration. These flaky precipitates, however, show no dichroism, and after long standing assume a violet colour. As a whole, the test is not very exact and reliable.

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[174] Heidenhain, Pflüger's Archiv, 1875, Bd. 10, P. 537.

[175] Hoppe-Seyler, Handbuch der physiologischen und pathologisch-chemischen Analyse, 7 Aufl., P. 390.

3. *By Means of the Spectroscope.*—When the blood is fresh, the gastric filtrate may be examined directly. Both stripes of the oxyhæmoglobin can then be seen very distinctly. When *free* hydrochloric acid or a large amount of organic acids is present in the gastric contents, the oxyhæmoglobin is changed into hydrochlorate of hæmatin, and, as a rule (according to my experience), nothing is seen. This is due to the fact that only traces of hæmatin dissolve in water. To dissolve the hæmatin, we proceed (according to Fr. Müller[176] and H. Weber[177]) by placing the suspected gastric contents into a test-tube with a few drops of concentrated acetic acid, and extracting it with about one-fifth its volume of ether. If the fluid contains blood, very soon the ether assumes a brown colouration. This solution of hæmatin in ether shows the following absorption-lines: 1, in the red; 2, in the yellow; 3, between yellow and green; 4, between green and blue. The absorption-line in the red is the most distinct. To be sure, this line, according to H. Weber, is not decisive, in so far as the chlorophyll, which may be found in the vegetable matter, in the stomach as well as in the intestines, shows a similar line. Weber recommends, in order that we may be sure as to this point, that after treatment with alcoholic potassium lye, the blood-pigment be taken up from the ethereal extract in a watery alkaline solution and treated with ammonium sulphate. The liquid now becomes red in colour, and in the spectroscope we observe the spectrum of reduced hæmoglobin (two lines in the green). The spectrum of chlorophyll is not altered through this procedure.

Another method is to mix the filtrate with a few cubic centimetres of concentrated acetic acid and to extract with sulphuric ether. On account of the hæmoglobin or hæmatin which it takes up, the ether assumes a sort of Tokay wine colour. If this colour does not appear and the ether remains clear, and but slightly affected as to change of colour, there is, according to H. Weber, certainly no blood present. Often, however, the ether assumes also a greenish or a yellowish-brown colour (hydrobilirubin, chlorophyll, Weber), which makes the detection of blood difficult.

4. *Hæmin Test.*—A small sample of the filter residue is taken and carefully evaporated on a watch-crystal over a small flame; the dried residue is now scratched off and mixed with a very little finely-triturated common salt. The mixture is now placed on a slide, and one or two drops of glacial acetic acid are added. This is carefully heated till the first

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[176] Fr. Müller, in Seifert-Müller, Klinisches Taschenbuch, Wiesbaden, Bergmann.

[177] H. Weber, Berliner klinische Wochenschrift, 1893, No. 19.

appearance of bubbles, and then is left to cool. With a high-power lens we now find the well-known blackish-brown rhombic crystals of hæmin (hydrochlorate of hæmatin) lying usually in little red-brown clods. According to the investigations of H. Weber, which I am able to confirm, the hæmin test, ordinarily so decisive, is not always of value for the gastric contents, because it may turn out negative even when blood is undoubtedly present.

5. *Guaiac Test (Almén's Test, van Deen's Test).*—A small sample of the gastric contents is mixed with 1 cubic centimetre of freshly prepared tincture of guaiac, and a like amount of Hühnerfeld's mixture (glacial acetic acid 2.0, aq. dest. 1.0, ol. terebinthin. et spirit. vin. rectif. of each 100.0), which mixture is thoroughly shaken. If blood is present in the gastric contents, the mixture assumes a bluish tinge after a short time. Carried on in this manner the guaiac test is not reliable, because a great number of food-materials (vegetable matter, milk) and also bile, saliva, pus, and likewise inorganic materials given as drugs, may give a like reaction. According to the investigations of H. Weber,[178] the test is reliable in the following modification.

A sample, as copious as possible, is diluted with water,* with the addition of about one-third as much glacial acetic acid, and is then shaken up with ether. After sedimentation, several cubic centimetres of this acid extract are poured off, and treated with about ten drops of tincture of guaiac and about twenty or thirty drops of resinous oil of turpentine. If blood is present, the mixture becomes bluish-violet; if blood is absent, it becomes reddish-brown, with perhaps a suggestion of green. The blue pigment may be extracted after addition of water with chloroform or, better (according to my experience), with amyl alcohol.

[J. Dutton Steele,[179] Philadelphia, has made several hundred separate tests for occult blood, whereby he used, besides the oil of turpentine, hydrogen peroxide; the other test used by Steele was the aloin test of Klunge. According to Steele's experience, hydrogen peroxide gives a more prompt and more delicate test with the guaiac than does the ozonised oil of turpentine, although the latter acts much better than the aloin test. This aloin test is made, according to Steele, as follows: a fresh solution of aloin is prepared by dissolving in 10 cubic centimetres of seventy per cent. alcohol as much aloin as can be taken on the tip of a spatula. The ethereal-acetic extract of the fæces is prepared in the same way as in the guaiac test. To

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* According to my experience, dilution with water is necessary only when there is much blood present, as, for instance, when the gastric contents are very black or like coffee-grounds, or when the stools are tarry.

[178] H. Weber, *l.c.*

[179] Pennsylvania Medical Journal, February, 1905.

2 cubic centimetres of the ethereal extract an equal quantity of the aloin solution is added and then 10 to 15 drops of either ozonised oil of turpentine or hydrogen peroxide is dropped in drop by drop. Here the turpentine acts much better and more delicately than does the hydrogen peroxide. If blood is present, the lower half of the fluid in the test tube will turn a rich cherry-red after standing for a short time, while in the absence of blood it will remain a light yellow.]

6. *By Means of the Iron Test.*—This is carried out according to Korczynski and Jaworski[180] as follows: a small quantity of the dark sediment to be tested is put into a small porcelain dish, mixed with a small quantity of potassium chloride and a few drops of concentrated hydrochloric acid, and then heated slowly over a small flame. If necessary, hydrochloric acid is added until the dark colour of the sediment entirely disappears. After the chlorine has completely escaped, one or two drops of a diluted potassium ferrocyanide solution are added, and if blood pigment is present, a decided blue colour (Prussian blue) appears. According to my experience, this test is very *sensitive,* provided it is certain that the patient has not taken any preparation of iron.

Pus in the gastric contents may be detected with certainty by the microscope. It also gives a blue colour with tincture of guaiac by itself (Vitali, Brücke, K. Brandenburg).

Diagnostic Significance of Occult Blood in the Stomach.

By occult hæmorrhages in the stomach or intestine are meant those bleedings which cannot be detected by the most careful macroscopic investigations. In the first place they occur, of course, when acid is present, but also when it is absent, if an intimate and long-continued admixture of slight gastric bleeding with the gastric contents or the fæces has existed. One cannot, however, speak of occult hæmorrhage when particles of mucous membrane or fresh blood, even if in minimal quantity, are discovered by careful inspection of the sample, which may be examined more carefully when it is on a black plate.

Investigations by Kochmann and myself[181] on a great number of patients suffering from gastric affections have shown that occult hæmorrhages are absent altogether in some gastric diseases; in others, however, are present sometimes regularly and sometimes intermittently. We never found occult blood in achylia gastrica (although artificial hæmorrhage may frequently be caused by forcible expression) and never in atonies and neuroses. It was found in variable constancy and frequency in gastric ulcers and in benign stenoses of the pylorus as long as stagnation occurs, but not, when only supersecretion is present. Occult hæmorrhages occur most frequently in gastric carcinoma, but in rare cases they may be absent. Thus the following diagnostic principles may be established: when occult gastric hæmorrhages or occult hæmorrhages in the fæces are constantly absent, if hydrochloric acid is absent and motility is well maintained, the disease is in all probability not gastric carcinoma. On the other hand, in the absence of hydrochloric acid, and in the presence of stagnation of the gastric contents and of lactic acid fermentation, a pronounced guaiac reaction speaks more for carcinoma than do the first three alone. Occult gastric hæmorrhage has no significance for the early diagnosis of a carcinoma of the stomach.

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[180] Korczynski and Jaworksi, Deutsche medicinische Wochenschrift, 1887, No. 47 and 49.

[181] Boas and Kochmann, Archiv für Verdauungskrankheiten, 1902, Bd. 8, Heft 1 and 2.

Abnormal Fermentative and Putrefactive Products in the Gastric Contents.

In high degrees of stagnation of the gastric contents there is found a whole series of fermentative products, which must in great part if not in whole be referred to the action of bacteria. Although the producers of fermentation as well as their products are known to us only to a small extent, there can be no doubt that they play an extraordinarily important part in the symptom-complex of diseases of the digestive apparatus.

Of most interest are the carbohydrate and the proteid decompositions. We have already considered as products of the former, lactic, butyric, and acetic acids; and furthermore, butyric acid fermentation leads probably (G. Hoppe-Seyler) to the formation of hydrogen. Here also should be mentioned the development of alcohol and carbonic acid under the influence of yeast-fermentation. Ehret[182] detected, besides alcohol and CO_2, also aldehyde, acetic acid, and formic acid in the stagnant contents containing hydrochloric acid, and he ascribes these fermentations to the presence of sarcinæ (page 239). As decomposition products of albumen are known ammonia and hydrogen sulphide, the occurrence of which, especially of the latter, under abnormal conditions, has been rather exactly investigated.

To a certain degree, the limit of which has not as yet been definitely established, these substances belong to the normal fermentation products; but, when they occur in large quantities, they certainly cannot be considered as anything but abnormal. This applies specifically, as has already been mentioned, to lactic acid, hydrogen, and hydrogen sulphide, which, as fermentation products, occur normally in the gastric contents, only in traces or not at all.

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[182] Ehret, Mittheilungen aus den Grenzgebieten der Medicin und Chirurgie, 1897, Bd. 2, P. 744, and 1898, Bd. 3, P. 579.

Rosenheim[183] and Strauss[184] have proved as the result of investigations, that as far as ammonia is concerned, it occurs normally in small quantities (0.15 to 0.17 per millé). Carbonic acid in small quantities may also be considered as among the normal substances present, as it is formed from sodium carbonate on admixture of the intestinal juice with the gastric juice, and thus may easily reach the stomach.

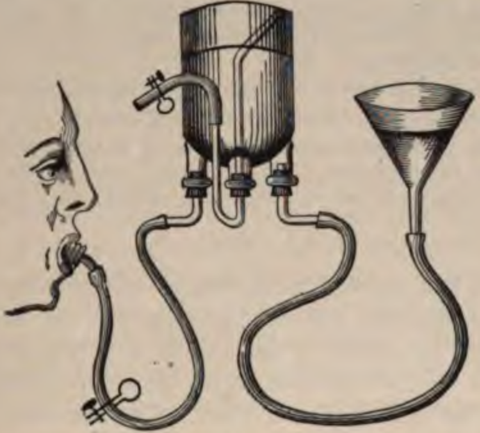

Fig. 28.—Hoppe-Seyler's Apparatus for Collecting Gases of the Stomach.

Our knowledge concerning the fermentation occurring in the stomach has been enriched materially of late through the investigations of G. Hoppe-Seyler,[185] Kuhn,[186] McNaught,[187] Strauss,[188] Talma,[189] Ehret and, I may add, of my own.

The author first named has planned, for the collection of the gases of the stomach, a well-adapted apparatus easily put together (see Fig. 28). A Wulffian bottle contains rubber stoppers in its three openings, the central one of which is perforated with a thin glass tube that extends to the bottom of the vessel, and the stoppers on either side are perforated with short, wide tubes bent at an angle. The thin glass tube is bent in the bottle in such a manner that it reaches the periphery of the bottom, so that on inclination of the bottle the gas may be taken up almost entirely; while outside the bottle the tube has two curves at right angles, so that, by means of a rubber tube provided with a clamp, gas may be removed at convenience. To one of the lateral wide glass tubes is attached a stomach-tube by means of a rubber tube provided with a clamp, and the other is attached to the funnel by means of a long rubber tube.

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[183] Rosenheim, Centralblatt für klin. Medicin, 1892, No. 39.

[184] Strauss, Berliner klin. Wochenschrift, 1893, No. 17.

[185] G. Hoppe-Seyler, Deutsches Archiv für klin. Medicin, 1892, Bd. 50, P. 82-100.

[186] Kuhn, Zeitschrift für klin. Medicin, 1892, Bd. 21, P. 572.

[187] McNaught, British Medical Journal, 1890, No. 1522.

[188] Strauss, Zeitschrift für klin. Medicin, 1894, Bd. 26 and 27.

[189] Talma, *ibid.*, 1899, Bd. 35, P. 542.

For taking up the gas the bottle is now filled with water through the funnel. The apparatus is now so placed that the flask hangs upside down, somewhat higher than the mouth of the patient. After the stomach-tube has been introduced, the funnel is lowered and the stomach-contents flows into the bottle, so that the bottle comes to contain a liquid which is saturated with about the same amount of gas as the stomach-contents. After a certain quantity of gastric contents have been drawn off, gas-bubbles come along with the liquid and collect in the upper part of the bottle. The imprisoned gas is now liberated through the central tube, and after its collection may be investigated chemically.

We can proceed according to Fr. Kuhn more simply, and in a manner better adapted for practical purposes, by filling a Fiebig's tube (such as is used in estimating sugar in the urine) with ten cubic centimetres of the fresh gastric contents, and putting this in the incubator. Fill to the zero point of the scale when the air in the apparatus has escaped and the liquid has reached body temperature, which is the case in one hour.

In absence of a regular fermentation tube, there can be used, as Riegel[190] recommends, the contrivance suggested by Moritz and Ewald for the estimation of sugar; it consists of a test-tube provided with a perforated rubber stopper, a right-angled glass tube running through the hole. After the test-tube has been filled to the rim with gastric contents, it is closed with the rubber stopper carrying the glass tube. The latter now fills with the expelled fluid, the whole apparatus becoming air-tight. This is now placed upside down in a beaker. Positive conclusions for diagnosis have not as yet been reached through these investigations.

The occasional presence of inflammable gases has been established since the early observations of Waldenburg,[191] Popoff,[192] Friedreich and Schultze,[193] Ewald,[194] and McNaught;[195] but the above-mentioned investigations of Hoppe-Seyler and Kuhn first made clear that inflammable gases, especially hydrogen, occur with extraordinary frequency in gastric diseases associated with stagnation. As natural constituents, besides hydrogen, Hoppe-Seyler found carbonic acid, and Kuhn, in addition to this, also found nitrogen and marsh-gas. The abnormal formation of gas is very common in mixtures remarkable for a high degree of hydrochloric acid.

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[190] Riegel, Die Erkankungen des Magens, I Teil, P. 140.

[191] *Cit.* by F. Kuhn, *l.c.*

[192] Popoff, Berliner klin. Wochenschrift, 1870, No. 38.

[193] Schultze, Berliner klin. Wochenschrift, 1874, No. 27 and 28.

[194] Ewald, Archiv für Anatomie und Physiologie, 1874, P. 217.

[195] McNaught, *l.c.*

Besides the gases named, the formation of *hydrogen sulphide* was occasionally observed in the body under pathological conditions by Senator,[196] Betz,[197] and Emminghaus,[198] with the subsequent absorption of this poisonous gas. As I demonstrated several years ago,[199] the formation of H_2S in the stomach, if stagnation is present, is by no means rare. Since the publication of my article in the year 1892, I have paid special attention to the presence of H_2S in benign ectasias, and have since observed it as an extraordinarily common occurrence. Zawadski[200] could confirm this observation in four cases of motor insufficiency. Strauss[201] reported a case of hydrogen sulphide gas fermentation in the stomach, but in this case the origin of the gas is doubtful, as it referred to a stenosis of the intestine. Strauss designates the bacterium coli as the cause of the hydrogen sulphide fermentation. Hydrogen sulphide fermentation occurs especially frequently in old cases of benign stenosis, which were not treated at all or were treated irrationally, while it was found very rarely in carcinoma with lactic acid fermentation.[202] I have observed hydrogen sulphide fermentation in both low and high degrees of hydrochloric acidity. It is demonstrated partly through its specific odour, and partly through the change in a strip of paper saturated with an alkaline solution of sugar of lead, which is to be suspended in a well-corked vessel containing the gastric contents. Rubin[203] recommends, as a reliable reagent, the administration of bismuth salts. If H_2S is present, the black crystals of bismuth sulphide can be found by means of the microscope.

The formation of H_2S in the stomach is undoubtedly to be regarded as bacterial. Thus Dauber[204] has been able to demonstrate partly in normal cases, partly in cases of motor insufficiency, thirteen kinds of bacteria which are vigourous H_2S formers, and ten varieties that form it moderately or slightly. The most of the producers of H_2S were found in cases of motor insufficiency of high degree. The bacteria cultivated by Dauber exhibited extraordinary resistance against hydrochloric acid. From all these investigations the fact, significant practically, may be drawn, that the formation of abnormal fermentative products is inhibited, to be sure, by hydrochloric acid, but only when the motor power is normal. With the accumulation of stagnant ingesta the presence of an abundant amount of hydrochloric acid does not interfere with the formation of liquid or gaseous decomposition products.

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[196] Senator, Berliner klin. Wochenschrift, 1868, P. 254.

[197] Betz, Memorabilien, 1864, P. 145, and 1869, P. 1.

[198] Emminghaus, Berliner klin. Wochenschrift, 1872, No. 40.

[199] Boas, Deutsche medicinische Wochenschrift, 1892, No. 49.

[200] Zawadski, Centralblatt für innere Medicin, 1894, No. 50.

[201] Strauss, Berliner klin. Wochenschrift, 1896, No. 18.

[202] Boas, Centralblatt für innere Medicin, 1895, No. 3.

[203] Rubin, Wiener medicin. Wochenschrift, 1901, No. 9.

[204] Dauber, Archiv für Verdauungskrankheiten, 1897, Bd. 3, Heft 1 and 2.

As characteristic products of the decomposition of albuminous bodies we recognise the *acetones*. After v. Jaksch[205] had repeatedly demonstrated acetone in the gastric contents, Lorenz[206] took up the subject more closely and could repeatedly demonstrate acetone also in the distillate of the gastric contents, especially in dilatation of the stomach, the amount of acetone occasionally even exceeding that in the urine. In other cases of gastric diseases, acetone was demonstrated also in the distillate of the excrements. The presence of acetone in the stomach-contents has been refuted by Penzoldt[207] and Savelieff.[208]

Important facts concerning other products of the chronic *putrefaction of albumen* in the gastro-enteric canal have not as yet been ascertained, although without doubt these play a prominent rôle in the symptomatology.

The frequently observed poisonings due to ingestion of tainted meat, sausage, fish, and mussels depend on the introduction into the gastro-enteric tract of certain toxic bases, such as have been isolated, particularly by Brieger, Vaughan, Ehrenberg, and others from a series of tainted or poisonous foods. It will be the object of further investigations to isolate, according to the methods planned especially by Brieger,[209] bases formed in the course of the putrefaction associated with diseases of digestion, whereby, no doubt, new paths will also be opened in diagnosis.

Microscopic Investigation of the Gastric Contents.

The microscopic investigation of the gastric contents and the vomitus may sometimes offer valuable diagnostic information, on which account it ought never to be neglected. Thus one can review the constituent parts of the secretion during fasting, and then the alterations which the ingesta, under the influence of digestion, bring about in the stomach.

(*a*) *The secretion on fasting* (so far as it is free of food) consists normally of gastric juice mixed with mucus and saliva, and shows a series of microscopic elements, that may be readily distinguished as belonging to the latter two substances. Besides, there occur regularly food-remnants, vegetable and elastic fibers, muscle-fibrillæ, starch-corpuscles, fat-globules, and crystals of fatty acids, also leucocytes and *cell-nuclei* in larger or smaller numbers, which are due in part to the action of the gastric juice on the mucus-corpuscles or on the epithelial cells of the gastric mucous membrane. This process may be easily demonstrated experimentally by adding dilute HCl to the neutral or alkaline gastric mucus, when numerous cell-nuclei appear.

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[205] v. Jaksch, Zeitschrift für klin. Medicin, 1884, Bd. 8, P. 36.

[206] H. Lorenz, *ibid.*, Bd. 19, P. 19.

[207] Penzoldt, Deutsches Archiv für klin. Medicin, 1884, Bd. 34.

[208] Savelieff, Berliner klin. Wochenschrift, 1894, No. 33.

[209] Brieger, Ueber Ptomaine, Berlin, 1885, I Theil, P. 14 u. f. There also is to be found an extensive bibliography.

A frequent constituent, especially of the fasting secretion, is represented by the *snail* or *spiral cells* first described by Jaworski[210] (Fig. 29). We find these finely moulded products not in rare cases, as he supposed, but quite constantly, more or less clearly pronounced, in all *fasting* gastric contents containing HCl. Especially characteristic, as I have observed, are the small sago-granule-like mucous plugs, which, occurring almost constantly in the acid gastric secretion, collect on the bottom of the vessel.

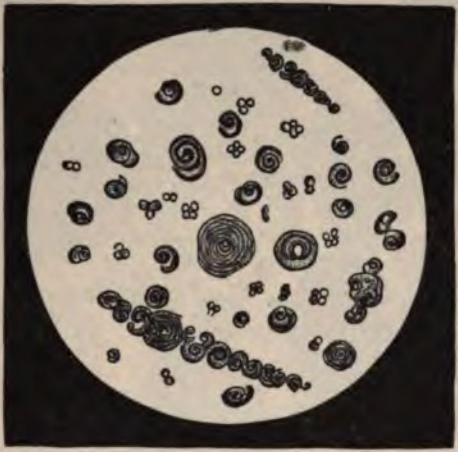

Fig. 29.—Spiral Cells from a Flake of Mucus from the Fasting Gastric Contents, between which are Cell-nuclei in the Form of Tetrads. (Personal observation.)

The cells named, according to the investigations of Tellering,[211] and as substantiated in my laboratory by P. Cohnheim, consist in nothing else than myelin altered by HCl, for we can induce the formation of these snail-cells on any specimen of bronchial or pharyngeal mucus to which sufficient HCl has been added. They occur partly singly (and, if so, are of considerable size), or in large groups of small-sized bodies; or may occur in the form of cells lying in apposition on threads resembling strings of pearls. In some formations the snail-form is very strictly preserved, while in others, usually large forms, the cells are more or less irregular. They disappear on long standing of the gastric contents, and one finds only cell-nuclei and masses of detritus. They can, however, be preserved for several days for demonstration if the gastric contents are made alkaline.

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[210] Jaworski, Münchener medicin. Wochenschrift, 1887, No. 33, and Verhandlungen des Kongresses für innere Medicin, 1888.

[211] Tellering, Inaug.-Diss., Bonn, 1895.

As far as I can see, since these formations may also occur in the acid chyme, especially when the latter accumulates much gastric or pharyngeal or bronchial mucus, a diagnostic significance cannot be ascribed to them.

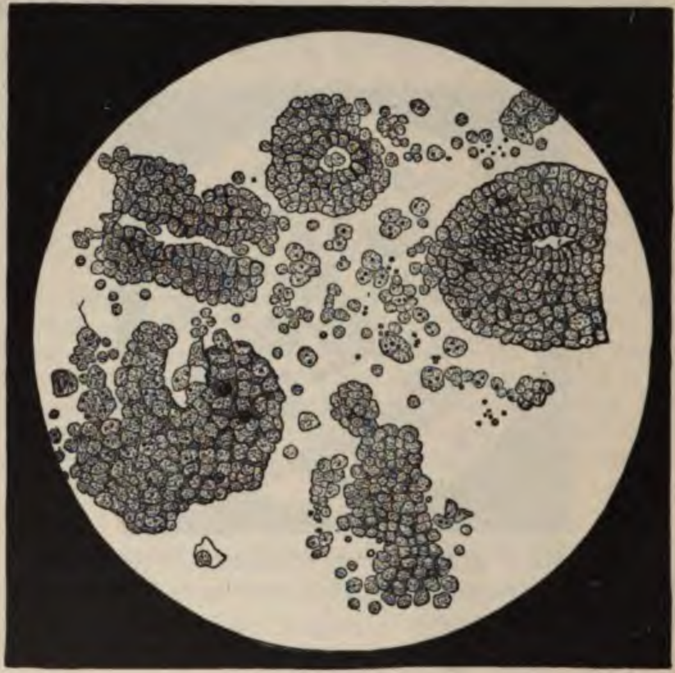

Fig. 30.—Fragment of Tubules of Glands of the Stomach.
(Personal observation.)

Now and again, also, desquamated epithelium, as well as fragments of *glandular tubules,* appear in the fasting stomach (Fig. 30), an occurrence not so rare as would appear from the description of others. Many times I have observed distinct fragments of glandular tubules, the texture of which could be determined with certainty. In all these cases the nuclei especially were distinct, while the protoplasm only became recognisable when a weak alkali was added. Not rarely did I see the well-known goblet-cells, sometimes single, sometimes more (two or three together). Frequently I observed also a few cells with the appearance of the parietal cell.

Bacilli and micrococci of various sorts never fail to occur in the fasting gastric juice. In more than a hundred microscopic examinations of the contents from the fasting stomach, I have never missed finding the leptothrix. Yeast-cells, partly singly, partly lying together in clumps, may also occur under normal conditions. Twice I found fungi in strongly acid contents (one case of phthisis pulmonalis, and one of anæmia). Kellogg[212] in 7000 examinations of the stomach-contents found mould-fungi 457 times, and that in 32 cases of simple dyspepsia, in 176 cases of hyperchlorhydria, and in 249 cases of pronounced hyperchlorhydria. The most frequent forms were oidium lactis, aspergillus fumigatus and flavescens. Einhorn,[213] who has recently been studying the occurrence of mould-fungi in the gastric contents, found these in the stomach contents in four cases (three cases of hyperchlorhydria, one case of gastritis chronica with erosions of the stomach) (Fig. 31). He thinks it possible that the mould-fungi

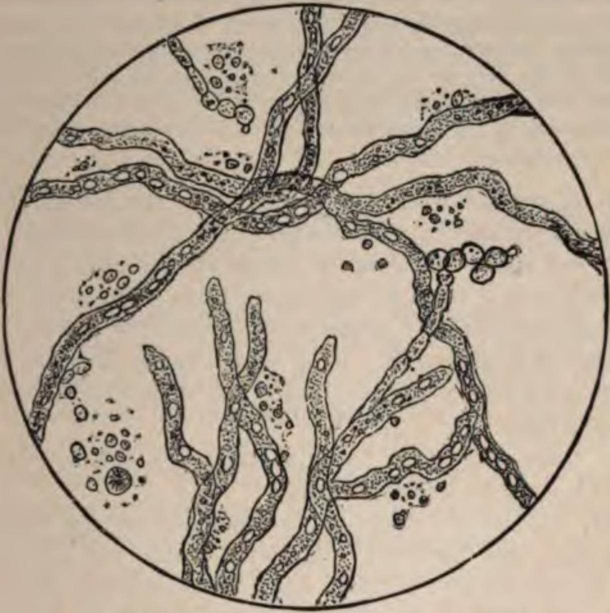

Fig. 31.—Mould Fungi in Gastric Contents. (After Einhorn.) Enlargement, 420.

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[212] Kellogg, New York Medical News, 21 July, 1900.

[213] Einhorn, Deutsche medicinische Wochenschrift, 1901, No. 37.

stand in connection with the affections named, or at least certainly aggravate them. Especially would this be the case, were the colonies to insinuate themselves into a fold of the surface of the mucous membrane, so that the onward flow of the chyme would not carry them along.

Petterson[214] also found a short time ago, in a case of perforated ulcus ventriculi, a sort of fungus in the contents of the peritoneal cavity, which was recognised as the *dematium pullulans de Bary*.

Rudnew[215] found in the mucosa and submucosa of the lesser curvature small, flat tumours of granulation-tissue which were broken through by numerous plant-filaments; he was dealing with penicillium glaucum. Kundrat[216] observed, in a case of universal favus, innumerable punctiform losses of substance in the mucous membrane. Yellowish exudate masses were found on the infiltrated base, and on the posterior wall of the pylorus was a membrane as large as a half dollar, which was tightly adherent to the reddened mucous membrane. The examination of the same showed the presence of the favus fungus. In one case, Rosenheim[217] found thrush fungus in the vomitus.

In several cases I could also demonstrate the butyric acid bacillus (clostridium butyricum) in the secretion of the fasting stomach; it is characterised by the known bluish-black colour when iodine is added, and its whetstone or lemon shape. In one case I found the butyric acid bacillus in the strongly hydrochloric secretion (fasting) of a patient with ectasia (pylorus stenosis after ulcer). In a large number of cases I found the bacillus with ends like a button (drum-stick shape), discovered by Bienstock,[218] in the fæces.

Besides these, still other bacteria occur, the detailed description of which would become too circumstantial.[219]

Further, *crystals* are found in the sediment of the contents of the fasting stomach, and especially in those containing bile. Cholesterin plates and lucin balls are relatively commonly met with, the former being easily recognised by their characteristically nicked form and their microchemical behaviour, the latter by their shape and colour. Never, however, have I observed tyrosin needles. Twice in the neutral gastric contents I also observed crystals which, in their configuration (long rhombs), resemble basic magnesia phosphate. They dissolved on the addition of acetic acid.

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[214] Petterson, *ibid.*, 1902, No. 39.

[215] Rudnew, Virchow-Hirsch's Jahresb., 1867.

[216] Kundrat, Wiener medicin. Blatter, 1884, No. 49.

[217] Rosenheim, Pathologie und Therapie der Krankheiten der Speiseröhre und des Magens, 2 Aufl., P. 207.

[218] Bienstock, Zeitschrift für klin. Medicin, 1884, Bd. 8, S. 27.

[219] Compare Abelous, Recherches sur les microbes de l'estomac à l'état normal, Paris, 1889.

Naunyn also mentions a case of catarrh of the stomach, in which he found numerous oxalic acid crystals in the aspirated contents; they disappeared after treatment continued for some time.

(*b*) The microscopic examination of the *gastric contents* after the ingestion of food offers the possibility of studying, besides the mentioned organic and inorganic products, also the metamorphoses of the ingesta under the influence of digestion. Amylum, muscle-fibres, and fats undergo characteristic changes which are sometimes of use in making the diagnosis.

It is well known that the saccharification of the starches is markedly inhibited by an excess of acid, so that on the addition of iodine-potassium-iodide solution in these cases we find a large number of intensely blue-coloured starch bodies, as well as well-preserved elements exhibiting the characteristic of concentrated layers. When hydrochloric acid is absent, we find only seldom blue reaction, or none at all, and also clearly defined starch-granules are seen but rarely. If such is not the case, we must not neglect to examine the diastatic power of the saliva.

Besides starch-granules, we frequently find vegetable cells, also cellulose and vegetable fibres, each of them sharply characterised by the iodine reaction. (Fig. 32).

The appearance and the behaviour of the muscle-fibres vary very much, depending on the condition and the stage of the digestion. Well-preserved cross-striation in *numerous* muscle-fibres several hours after the ingestion speaks for a deficiency of the peptic power of the gastric juice, although in absence of cross-striation it may yet be insufficient, as the fermentative processes in the stomach institute a gradual maceration of the fibrillæ. We find muscle-fibres most commonly in the stagnant contents (see Fig. 34), which may occasionally exhibit, also, meat-particles visible to the naked eye. As soon as the cross-striation has disappeared, the fibrillæ appear as structureless clods, the origin of which can hardly be recognised.

Fat-globules and *crystals of the fatty acids* are also frequently found in the gastric contents; the former are recognised by their solubility in ether and through their staining with Sudan or scarlet-red, the latter by its solubility on heating and gradual reappearance on cooling. An abnormally excessive amount of fat-globules and needles of fatty acids is especially common in ectasia of the stomach, but is not confined to that disease, in which a stratum of fat can readily be observed swimming on the surface [of the contents].

Mucus and pus-cells, as well as numerous cell-nuclei, occur also in the gastric contents. Epithelium of the pharynx and of the œsophagus are by no means rare in the gastric contents.

Epithelial cells of the gastric mucous membrane are also found under normal conditions in the gastric contents, rather sparingly, to be sure, and requiring careful observation to detect; also glandular epithelial cells, some well preserved, some shrunken or broken up, finally some cloudy with granules. Of great practical importance is the occurrence of *nests of cancer cells* in the vomitus or in the contents, which have been observed by others (Ewald, Reineboth, and others), and also by myself. Their unequivocal presence is the surest criterion of a carcinoma of the stomach. Unluckily, it is almost always an accidental finding, and that in cases already well advanced.

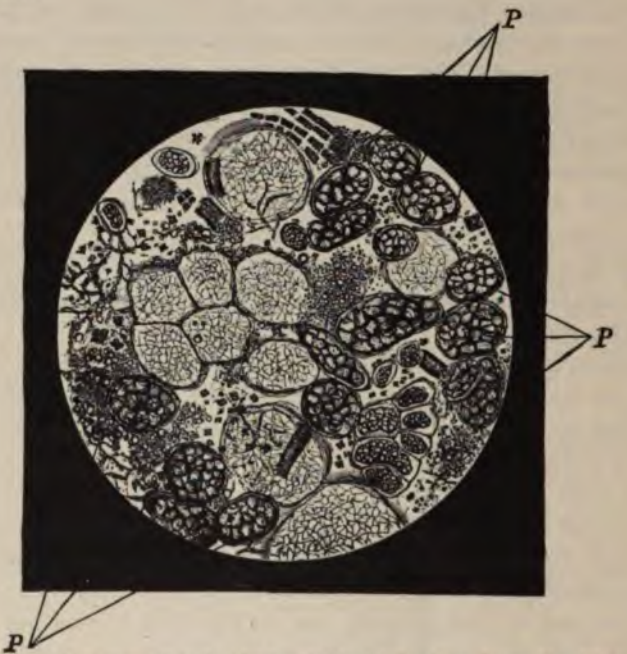

Fig. 32.—Different Kinds of Vegetable Cells (*P*) from the Stomach Contents in a Case of Ectasia. (Personal observation.)

Red blood-corpuscles are found in the vomitus or in the gastric contents, even in very slight hæmorrhages; in neutral or weakly acid contents they remain unaltered for hours, but when HCl is present they are destroyed. The diagnostic significance of blood in the vomitus or contents has already been stated.

Of fungi, there occur normally (in small quantity) the *yeast-fungi*. Sprue (oidium or saccharomyces albicans), originating for the most part in the oral cavity, is also met with. The occurrence of mould-fungi has already been mentioned. The question of the occurrence and significance of the bacteria, of which short mention has also been made, is very much complicated. Without doubt, in a large number of bacteria we have to deal with harmless bacteria which have been introduced with the food, or with parasites coming from the oral cavity, or with micro-organisms which have a rather favourable effect. It must be understood, on the other hand, that their number and the consequent abnormal changes may exert a harmful influence on the course of digestion.

As to morphology, de Bary, who especially has studied carefully the bacteria occurring in the stomach-contents, found in seventeen cases the following vegetable micro-organisms: sarcina ventriculi, fungi (oidium lactis, mucor-mycelia, indefinite forms), fungi (globular, long, chalara-shaped, neither of them productive of fermentation), leptothrix buccalis, bacillus amylobacter, and a form resembling the bacillus subtilis (see above) which de Bary has called bacillus geniculatus. This forms zigzag-shaped spores, and shortly after the bending show independent motility. The smallest are 4 to 5 μ long, and 0.5 to 0.6 μ broad. They are destroyed, according to de Bary and as I also frequently had occasion to observe, by 0.2 per cent. hydrochloric acid. It is, however, evident that the flora of the fungi of the stomach is not limited to those mentioned. On the basis of his investigations, de Bary[220] comes to the conclusion that the fermentative activities of the fungi as a factor in the production of dilatations and other gastric disturbances have been overestimated. Miller,[221] in addition to a series of other facts, has demonstrated the important truth, *that the fungi, after entering the stomach, are by no means destroyed by the hydrochloric acid, but in part pass into the intestine in a condition capable of further development.*

Of late years, Abelous, J. Kaufmann, Gillespie, and others have instituted thorough investigations on the bacterial flora of the stomach. Others have concerned themselves more with the study of individual forms of bacteria. On the whole, the knowledge obtained by these investigations is slight, as they have shown that the stomach shelters a great number of species of fungi of the most different kinds, which is not strange when we consider that the bacteria are carried to the stomach from the air and the saliva, as well as from food and drink. The unlikelihood of isolating specific bacteria from this complex mixture is apparent.

Nevertheless, it appears to me that the long, thread-shaped, immobile bacilli, first observed by myself, and later accurately described by Oppler,[222] and cultivated by Kaufmann and Schlesinger,[223] and which are found in especially large numbers in gastric carcinoma (in inacidity and stagnation), must be considered as the lactic acid bacillus, κατ' ἐξοχήν, *(Fig. 33). We must agree with Strauss in accepting a characteristic finding only when the bacilli fairly overwhelm the field. With this restriction, the demonstration of the bacilli named by me "thread-bacillus," especially as a supplement to the chemical demonstration of lactic acid, is not without diagnostic value.

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[220] de Bary, Archiv für experimentelle Pathologie und Therapie, Bd. 20, P. 243.

[221] Miller, *l.c.*, P. 250.

[222] Oppler, Deutsche medicinische Wochenschrift, 1895, No. 5.

[223] Kaufmann and Schlesinger, Wiener klinische Rundschau, 1895, No. 15.

As far as the other micro-organisms of the stomach are concerned, we have, as Minkowski[224] very correctly states, to consider the number rather than the kind of bacteria in making the diagnosis of an abnormal fermentation.

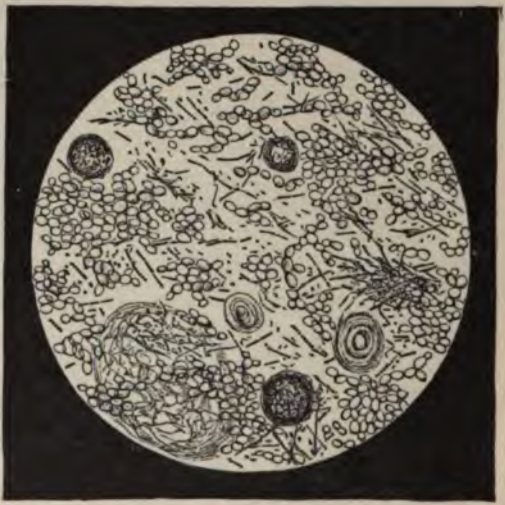

Fig. 33.—Yeast Ferment in the Stomach with the Simultaneous Presence of Thread Bacilli. (Personal observation.)

The disturbances which depend upon an abnormal accumulation of bacteria may be explained, according to Minkowski, in the following manner:—

1. Substances may be formed which irritate the gastric mucous membrane and bring on a catarrhal inflammation.

2. Gases may be formed in considerable quantities, which occasion subjective symptoms and increase the gastric insufficiency already present.

3. The fermentations may cause the production of substances which exert a toxic effect.

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* See also illustration in Special Part, page 578.

[224] Minkowski, Mitteilungen aus der medicinischen Klinik zu Königsberg i. Pr., Edited by B. Naunyn, Leipzig, 1888, P. 156.

4. In the fermentation of albuminous substances, alkaline products may develop which cause a neutralisation of the rest of the secreted hydrochloric acid.

5. Gastric fermentation may have a great influence on the function of the intestines.

Of great significance practically and diagnostically is the occurrence of *yeast fungi* and *sarcina ventriculi* in the gastric contents.

Yeast and sarcinæ may occur in small numbers in a variety of gastric diseases, but are observed in great number only in excessive stagnation of the gastric contents. Yeast and sarcinæ occur together very frequently.

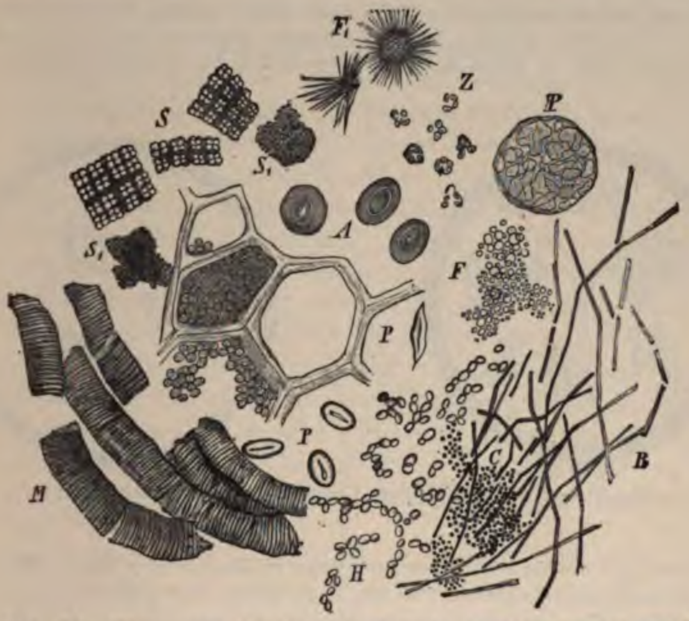

Fig. 34.—Microscopic Findings in Ectasia of the Stomach of High Degree. *A*, Starch-granules. *M*, Muscle-fibres. *S*, Sarcinæ in forms of bales. S_1, Degenerated sarcinæ. *P*, Vegetable cells and vegetable fibres. *F*, Fat-globules. F_1, Crystals of fatty acids. *H*, Yeast fungi. *Z*, Cell nuclei. *B*, Bacilli (Leptothrix). *C*, Cocci. (Personal observation.)

The appearance of yeast (Fig. 33) is very characteristic: the glistening, oval, double-contoured cells, frequently strung together in rows like beads, staining yellow with iodine, are not difficult to recognise.

The sarcinæ (Figs. 34 and 35) appear in the gastric contents in two different forms. First, in the common cotton-bale form, whereby the bundles appear sometimes larger, sometimes smaller, sometimes glistening brightly, sometimes brownish-yellow, which probably indicate the appearance of the organism at different ages. Second, in the form of irregular groups or cubically arranged bales, which consist of smaller individual organisms. All these varieties show distinctly the cellulose-reaction[*] which stamps their belonging to the vegetable kingdom. If sarcinæ occur in large numbers in the stomach, they will also be found in the fæces. They are then commonly impregnated by bile (Ehret).

Up to a short time ago the cultivation of the sarcinæ of the stomach was tried in vain. Falkenheim[225] succeeded in cultivating only the second species, and that only in hay-infusion. Oppler[226] was the first to isolate several forms in pure culture and in various media.

The cotton-bale sarcinæ (Fig 35*A*), consisting of groups of individuals of 8, 64, etc., grow in bouillon, gelatine, potato, agar-agar, especially well in hay-infusion to which two per cent. of grape-sugar has been added, at incubator temperature. In hay-infusion and bouillon it grows as a flaky sediment and a thick pellicle, while the liquid itself remains clear. On agar-agar, potato, and gelatine it forms lemon-yellow colonies, and liquifies the latter rapidly.

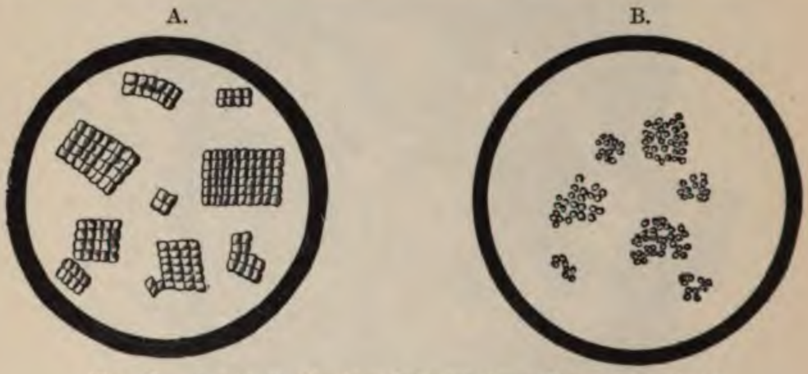

Fig. 35.—*A* and *B*, Sarcinæ Cultures from Stomach-contents.

The *second* form (Fig. 35*B*) shows individuals of much smaller size; the bundles are much more loosely bound and do not appear as bales of cotton. It grows on the same media, preferably on agar-agar and on hay-infusion with a two per cent.

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[*] We treat a sample of the specimen rich in sarcinæ with a large drop of the following solution:—

Chloride zinc	20.0
Iodide of potash	6.5
Iodine	1.3
Water	10.5

and put on the cover-glass only after several minutes have elapsed. All the starches appear deep-blue, the bales of sarcinæ a pretty violet-red.

[225] Falkenheim, Archiv für experimentelle Pathologie und Pharmakologie, 1885, Bd. 19.

[226] Oppler, Münchener medicinische Wochenschrift, 1894, No. 29.

solution of dextrose. The colonies on the solid media are whitish-yellow, and gelatine is liquified much more slowly than by the first form.

Only a few, and by no means conclusive, investigations have been made as regards the biologic action of the sarcinæ. Ehret[227] found that the sarcinæ, in the test-tube, exert a natural influence on the production of hydrochloric acid, but bring about no gas-formation. He blames altered conditions of growth in the cultivation for the persistence of absence of gas-formation. Also Coyon[228] found in vitro only slight fermentation on addition of peptones as well as of carbohydrates. The question needs renewed and careful investigation. Doubtless the sarcinæ play some part in gas-formation, as they have disappeared when this stops.

The *diagnostic significance* of the sarcinæ was formerly much overrated by Bamberger and others. The presence of sarcinæ is not really specific of any disease, and they are found, with few exceptions, only in stagnation with presence of hydrochloric acid (it does not matter whether hypochlorhydria or hyperchlorhydria), but much more rarely in stagnation with achlorhydria or presence of lactic acid; indeed with the transformation of the one into the other form the sarcinæ may disappear within a few days. In case no stagnation is present, sarcinæ are either entirely absent or are formed only in exceedingly few and degenerated specimens.

Very commonly countless species of bacteria are found with these micro-organisms, in which case either putrefactive processes on the mucous membrane (for example, ulcerated carcinoma) or, on the other hand, a stagnation of the contents in the wake of an obstruction may be present. It is evident that the absence of the characteristic fungi does not exclude a motor insufficiency.

In the œsophagus and in the gastric contents, as in the fæces, **infusoria** and especially flagellates are often found. The first finding was made by Skaller[229] in my policlinic, in the stools of an individual with carcinoma of the œsophagus, in which tumour in all likelihood they were primarily located. A similar observation was made a short time afterwards by Strube[230] in a case of carcinoma of the cardia. In both cases trichomonas forms were present. In the stomach itself monads were found by Hensen[231] and P. Cohnheim,[232] in part trichomonads and in part megastome forms.

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[227] Ehret, Mittheilungen aus den Grenzgebieten der Medicin und Chirurgie, 1897, Bd. 2.

[228] Coyon, Compt. rend. soc. biol., 51, S. 967-970, ref. nach Maly's Jahresbericht für Thierch, 1900, S. 384.

[229] Skaller, Berliner klinische Wochenschrift, 1898, No. 25.

[230] Strube, *ibid.*, No. 32.

[231] Hensen, Deutsches Archiv für klinische Medicin, 1897, Bd. 59.

[232] P. Cohnheim, Sitzung des Vereins für innere Medicin of May 12, 1902, P. 450

A similar discovery was made by Zabel[233] in a case of papilloma developing upon a carcinomatous basis.

According to Cohnheim, the presence of the specified flagellates has a diagnostic significance, as they are indices of the presence of an ulcerating carcinoma not situated at the pylorus. They have nothing to do with the ætiology of the carcinoma of the stomach.

As in the fasting stomach, there occur at times, also, after the ingestion of food, formations resembling leucin, and that also when there is no recurrence of bile. I have seen these, especially, in stenosis of the pylorus and consecutive ectasia with fermentative and putrefactive processes in the stomach, and in one case I isolated the pure leucin. It was also possible to obtain tyrosin in moderate quantities, according to the procedure of Hlasiwetz and Habermann.

In the alkaline vomitus of a chlorotic girl, Eichhorst[234] found crystals of ammonio-magnesium phosphate (coffin-lid). I also have seen in two cases of ectasia the same forms of crystals, and in two other cases, as already mentioned, crystals of magnesium phosphate.

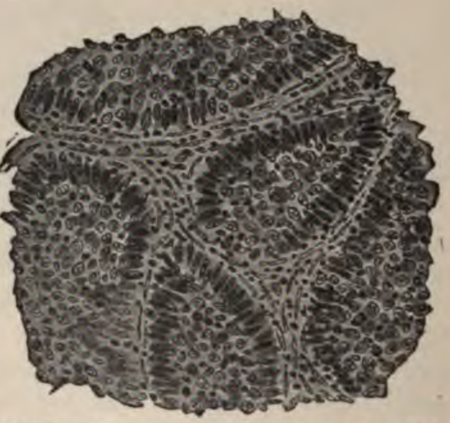

Fig. 36.—Medullary Carcinoma of the Stomach. Particle of tumour obtained by expression of gastric contents. (Personal observation.)

Of great significance is the finding of particles of tumours in the gastric contents, and this is by no means so rare as was formerly believed, and to which O. Rosenbach[235] first called attention. I myself have had several occasions to make a diagnosis from such particles of tumours (see Fig. 36), and the same has been the case with Rosenbach and Reineboth.[236] The last-named author, however, emphasises correctly that exulcerated particles found in the blood coagula do not always afford convincing pictures. It is plain, as Lubarsch[237] and Reineboth fittingly state, that an early diagnosis can scarcely ever be made on the basis of such a finding. Nevertheless, the finding may occasionally be of decided value.

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[233] Zabel, Archiv für Verdauungskrankheiten, 1901, Bd. 7, Heft 6.

[234] Eichhorst, Lehrbuch der physikalischen Untersuchungsmethoden, A Aufl., Bd. 2, P. 222.

[235] O. Rosenbach, Deutsches medicinische Wochenschrift, 1882, No. 33.

[236] Reineboth, Deutsches Archiv für Klin. Medicin, 1897, Bd. 58.

According to Samuel Fenwick,[238] pieces of the mucosa were also found in the *vomitus* in subacute gastritis [and tumour fragments in sarcoma].

Since I have paid more attention to the microscopy of the gastric contents, I frequently had occasion, in a considerable number of cases, to investigate small pieces of mucous membrane found in the contents, and have thus been able to substantiate the clinical diagnosis. Very commonly in expression of the gastric contents, already remarked, a bleeding occurs, which, it may be said in passing, is entirely insignificant; in many of these cases we may find, together with the blood, one or more particles of mucous membrane in the contents. In other cases there is no bleeding at all, although a careful examination of the contents shows one or several fragments of the mucous membrane. I never saw bad results follow such casting-off of the fragments of the mucosa, nor did I observe any change whatever in the condition of the patient; so that I do not regard such an occurrence, *which, of course, should not be purposely brought about by a rough handling of the stomach tube,* as important or, under such circumstances, as decisive for the diagnosis.

Exfoliation of the mucous membrane is particularly apt to occur in cases of chronic gastritis, as also in pronounced superacidity (without ulcer), and finally also in neuroses. I have thus obtained the impression, that many a mucous membrane may be decidedly softened so that a trivial injury or merely a simple pressure may bring about slight abrasions of the mucosa. The injuries are so unimportant and the pressure so insignificant that I cannot get rid of the idea that in a markedly softened and swollen mucosa, perhaps even through the act of defecation, similar abrasions may occur, which, of course, remain latent in the beginning.

As Einhorn[239] first observed and Pariser[240] recently confirmed, on lavage of the stomach such particles may be obtained in individual cases, and indeed constantly. Einhorn and likewise Pariser believe that this finding should be referred to a specific form of disease, characterised anatomically by the well-known *hæmorrhagic* erosions. The subjective troubles consist of severe pains after ingestion of food, falling off in weight, and weakness. The gastric contents show nothing characteristic. According to my view, however, no disease *sui generis* is present, but a symptom-complex, the constancy of which must first be proved by much material.*

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[237] Lubarsch-Martius, Ueber Achylia gastrica, Leipzig and Wien, 1897, page 161.

[238] Samuel Fenwick, The Morbid States of the Stomach and Duodenum, etc., London, 1868, P. 308. (Cited by Ebstein, Berliner klin. Wochenschrift, 1895, P. 71.)

[239] Einhorn, Medical Record, June 23, 1894, and Diseases of the Stomach, New York, 1897, P. 234 *et seq.*

[240] Pariser, Medicinische Revue, 1897, No. 1.

The *diagnostic* value of the microscopic investigation of the fragments of mucous membranes depends on the control which may cover the clinical observation of the case, and then on the likelihood of making the diagnosis accessible in clinically doubtful cases.

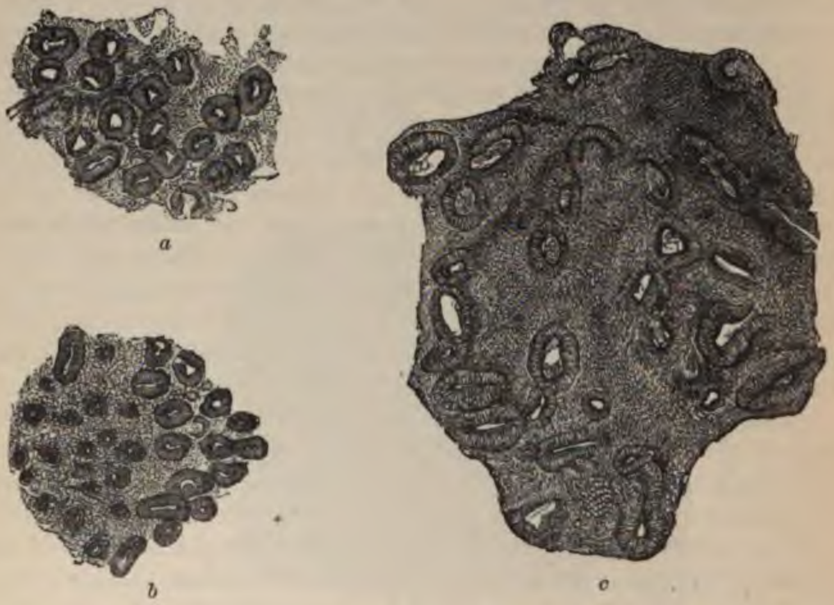

Fig. 37.—These three specimens show Small Fragments of the Mucosa, which, directly after they were obtained, were examined in physiologic salt solution and drawn. (Personal observation)

In the latter sense I might perhaps refer to the cases which are probably the greatest source of difficulty to us, namely, disturbances of digestion which simulate the clinical picture of the neuroses. The differential diagnosis between *neuroses* and *catarrh* is made considerably easier by the microscopic examination of a particle of mucous membrane.

These observations seem to give me a certain perspective for the more precise judgment of cases with insufficiency of secretion. The means hitherto employed for distinguishing stagnation-insufficiency from one due to

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* Compare Part II, page 470.

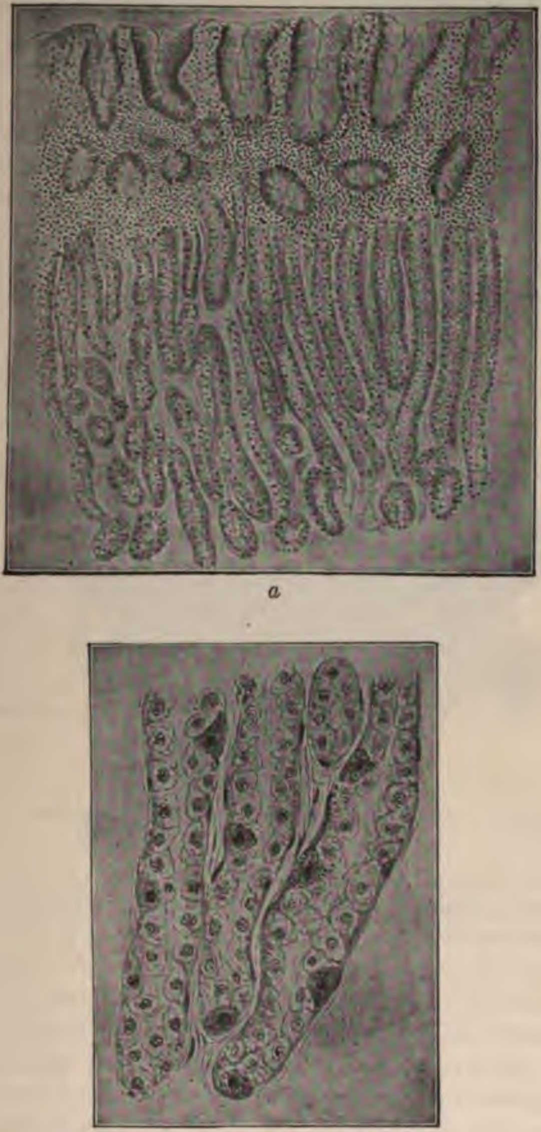

Fig. 38.—Normal Gastric Mucous Membrane from a Case of Atony of the Stomach (low-power microscope). In *b* are three tubules of glands with distinctly visible chief and parietal cells (high-power microscope). (Personal observation.)

organic changes in the glands are, as known, not satisfactory in all cases. This gap may occasionally be filled by a microscopic examination of exfoliated particles of the mucosa. Such a particle was obtained by me in a case of a patient ill with chronic enteritis and catarrh of the stomach, after the expression of a test-breakfast, that had been constantly free of HCl for weeks; this I examined partly fresh and partly hardened in alcohol. The examination showed a trivial interstitial gastritis, which conceded hope of restitution. In fact, a few weeks later an entirely normal secretion of HCl appeared, while the complaints of the patient (copious diarrhœa, pressure and fulness after eating) disappeared. Similarly, I found, in other advanced cases, remarkable for the duration of years, essentially more progressive changes in the mucous membrane.

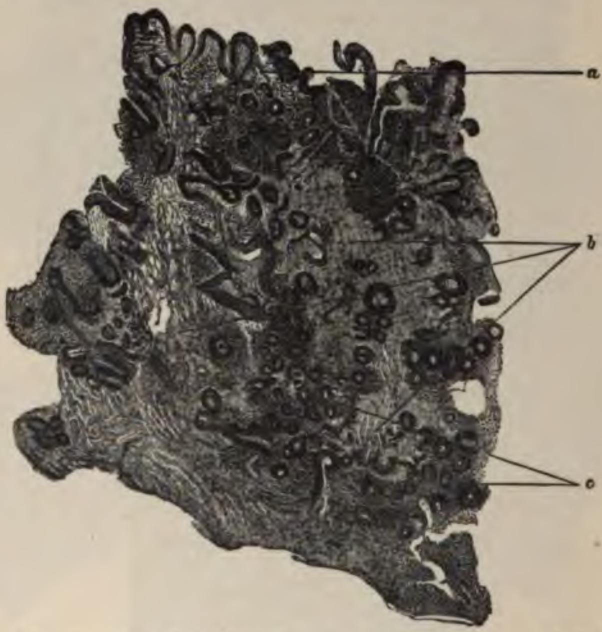

Fig. 39.—Gastritis Interstitialis. *a*, The free surface of the mucosa. *b*, Areas of destruction of glands. *c*, Comparatively intact zone. (Personal observation.)

The three illustrations (Fig. 37, *a*, *b*, *c*), show for instance pictures of a slight and a severe interstitial gastritis. While in *a* a scarcely essential increase of the interglandular tissue is to be observed, in *b* is to be seen a distinctly more advanced increase of connective tissue, which in *c* approaches actual sclerotic change. The parenchyma of the glands was intact in all three cases.

The best and most instructive pictures are obtained after hardening of the fragments in alcohol, embedding in celloidin or paraffine and staining of the sections in hæmatoxylin and eosin or alumcarmine. Congo stain also, which Stintzing[241] suggested, gives quite pretty pictures.

I choose from my specimens some typical cases, which will serve to illustrate the value of these investigations.

A piece of mucous membrane, from a case of atony of the stomach (Fig. 38, *a* and *b*), with superacidity, with normal condition of the mucous membrane, may be illustrated for comparison.

The commonest are cases of interstitial gastritis in more or less developed degrees. Fig. 39 shows the specimen of an advanced form of interglandular gastritis, in which the process has progressed in some places to obliteration of the glands (*b*). The glandular epithelial cells themselves are all intact where they are still distinct (*c*). During life in that patient, who was under my treatment for several years, there was constant absence of hydrochloric acid and considerable deficiency of the lab-zymogen and pepsinogen.

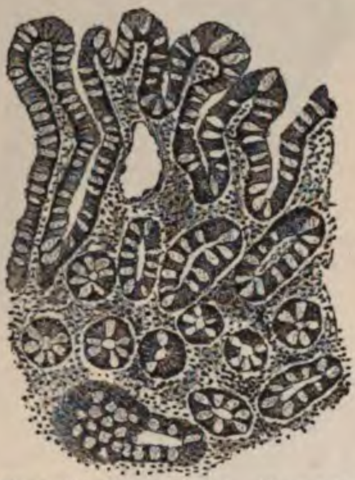

Fig. 40.—Gastritis with Goblet Cells. (Low-power microscope.) (Personal observation.)

On the other hand, Figs. 40 and 41 show the picture of a gastritis with marked (pathologic) production of goblet cells (not, as I formerly supposed, conversion of the glandular epithelium into mucus).

This form, recently studied, especially by A. Schmidt,[242] P. Cohnheim,[243] Hammerschlag,[244] Lubarsch,[245] Hári,[246] is found in pathologic processes of various kinds. In certain cases this form of gastritis is directly converted into atrophy of the gastric mucous membrane. In other cases they are observed simultaneously with atrophy. Such heterotopic formations are also seen in carcinoma of the stomach.

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[241] Stintzing, Sitzungsberichte der Gesellschaft für Morphologie und Physiologie in München, 1889.

[242] A. Schmidt, Deutsche medicinische Wochenschrift, 1895, No. 19.

Fig. 42 shows a hyperplastic gastritis. At certain places (*c*) one sees an advanced interglandular conglomeration of cells. This case also was characterised by constant absence of hydrochloric acid and ferments.

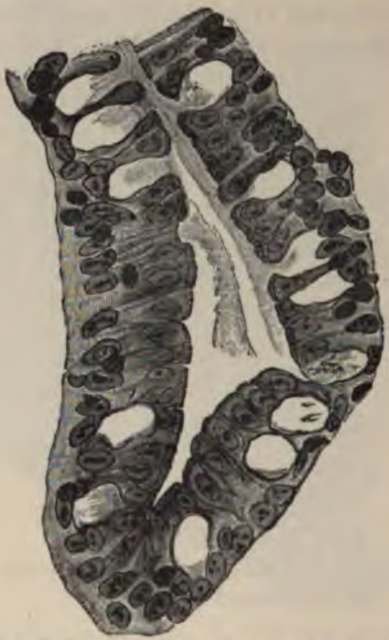

Fig. 41.—Isolated Antral Glands of the same case. (High-power microscope.) The goblet cells, as is to be seen from the picture, are partly filled with mucus. (Personal observation.)

In the next case (Fig. 43) there is a total atrophy of the glandular stratum, while the antral layer is in a state of marked proliferation. Clinically (according to investigations by P. Cohnheim and Hammerschlag, which I can confirm), this form is to be designated as a genuine atrophy of the mucous membrane.

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[243] Cohnheim, Archiv für Verdauungskrankheiten, 1895, Bd. 1, P. 274.

[244] Hammerschlag, *ibid.*, Bd. 2, P. 206.

[245] Martius, Ueber Achylia gastrica mit einem anatomischen Beitrag von Prof. O. Lubarsch, Leipzig and Wien, 1897.

[246] Hári, Archiv für mikroskopische Anatomie, 1901, Bd. 58.

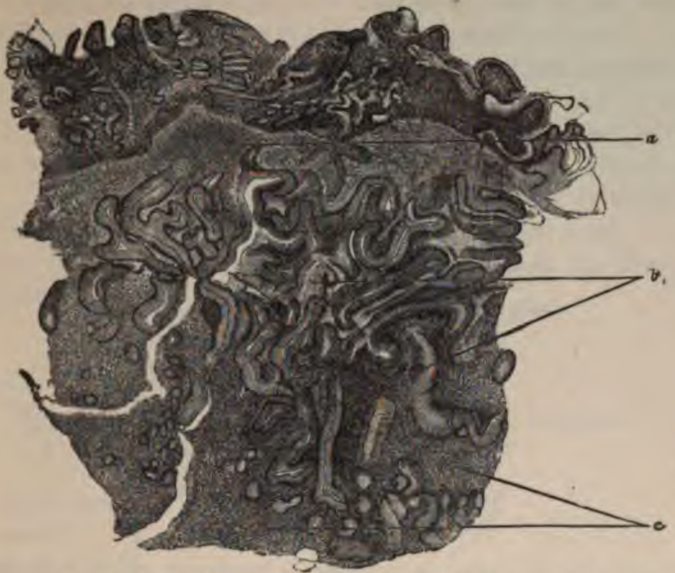

Fig. 42.—Gastritis Hyperplastica. At *b* the hyperplastic glandular tubules, at *c* marked interglandular infiltration, at *a* hæmorrhagic infiltrate. (Personal observation.)

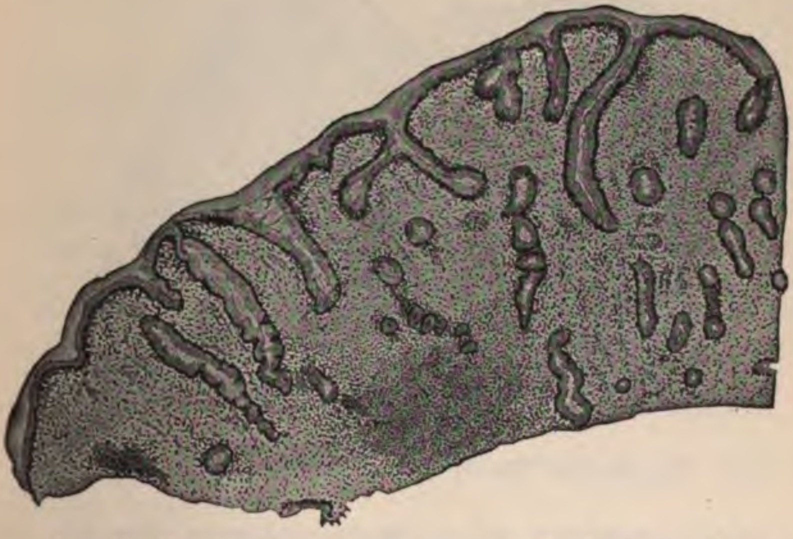

Fig. 43.—Gastritis Atrophicans. One sees in the figure scarcely anything except the hypertrophic antra, while the glandular region has wasted away, a few remnants excepted. (Personal observation.)

In Fig. 44 we have to do with an exquisite case of phthisis mucosæ ventriculi. The glandular structure has here been lost and is restricted to a slight remnant, and in one place is seen a large infiltration of leucocytes.

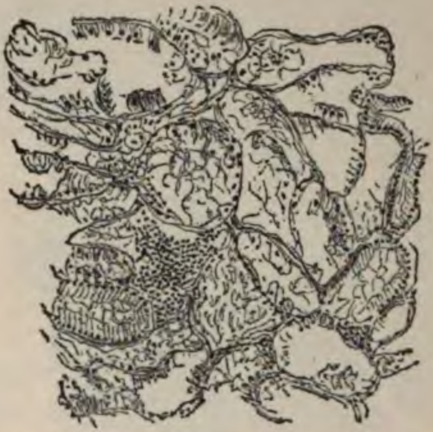

Fig. 44.—Atrophy of the Gastric Mucous Membrane. Phthisis mucosæ. (Personal observation).

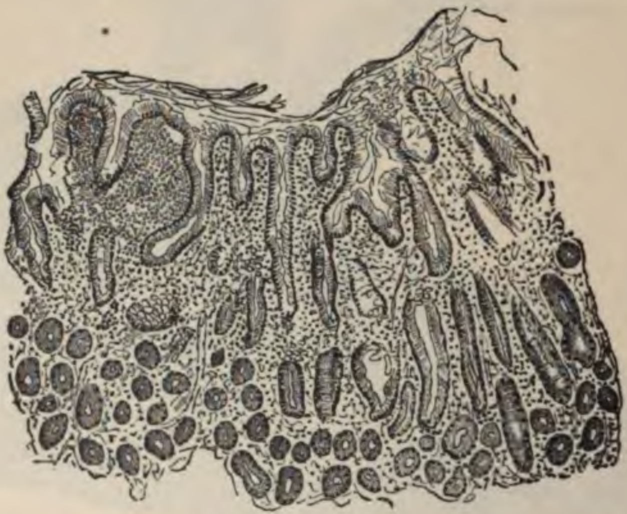

Fig. 45.—Gastritis Proliferans. (Personal observation.)

Finally I mention a case of gastritis proliferans (Fig. 45), distinguished chemically by a high degree of superacidity. One sees irregularly formed, markedly convoluted, and decidedly increased antral spaces, but with a well-preserved layer of glands. On the left side of the illustration above is a large accumulation of red blood-corpuscles.

The views concerning the significance of these examinations of fragments vary considerably. The objection raised on different sides (Lubarsch, Leuk, and others) that the examination of small fragments of mucous membrane does not yield a conclusive picture, is correct, as Hemmeter also contends. Nevertheless, the findings and the histologic investigation of pieces of mucous membrane (and here I agree with Hemmeter, in spite of the contrary opinion of the other authors), contribute to the clearing and the confirming of the diagnosis.

Synoptic Course of Examination of Gastric Contents.

1. *Macroscopic Examination.*

1. Appearance }
2. Quantity } of the gastric contents.
3. Odour }

4. Possible admixtures (blood, pus, bile, duodenal juice, mucus, saliva, gas formation, fragments of mucous membrane, particles of tumour or mucous membrane.

2. *Chemical Examination.*

1. Reaction of the gastric contents.

2. The presence of free hydrochloric acid or organic acid, demonstrated by dye-stuff tests (congo, tropæolin, dimethylamidoazobenzol).

3. Demonstration of free hydrochloric acid by the proper HCl reagents (phloroglucin-vanillin, resorcin).

4. Tests for lactic acid by means of Uffelmann's reagent.

5. In doubtful cases, extraction of the filtrate by shaking up with ether, and a second test by the named reagents for detection of acetaldehyde.

6. When butyric or acetic acid is thought to be present, test for these after shaking up with ether.

7. Estimation of the total acidity by means of decinormal lye.

8. Estimation of the total hydrochloric acid.

9. Estimation of the free hydrochloric acid, according to Mintz, or Mörner-Boas, or Töpfer.

10. Investigation for enzymes and zymogens (pepsinogen and pepsine, lab-zymogen and lab-ferment).

11. Test for the digestion of albumen.

12. Test for the digestion of carbohydrates.
13. Test for mucus, saliva, blood, bile, duodenal juice, etc.
14. Test for occult gastric hæmorrhage.

3. *Microscopic Examination.*

1. Condition of chyme (starch, meat, fat).

2. Abnormal constituents—fragments of mucous membrane (particles of tumours), epithelium, red blood-corpuscles, leucocytes, pus, Jaworski's spiral cells, crystals, sarcinæ and yeast, sprout- and mould-fungi, bacteria, protozoa; and that (*a*) in the contents of the fasting stomach, and (*b*) in the contents after a test-breakfast.

CHAPTER VII.

Diagnostic Significance of the Examination of the Urine.

Our knowledge concerning the condition of the urine in stomach diseases is, we regret to say, scanty, and almost exclusively casuistic. A proper semiotic significance has not as yet been ascribed to the examination of urine, but there exist, nevertheless, some noteworthy building stones, which might perhaps, in the future, be used successfully for diagnosis. We will set forth in the following the important deviations in the urine in gastric diseases. The following come under consideration:

The amount, the reaction, the specific gravity, the condition of the chlorides, the phosphates, the combinations of sulphur, the amount of nitrogen, the amount of peptic ferments, and finally the presence of abnormal constituents (albumen, peptone, acetone, diacetic acid, and the indigo derivatives).

1. *The Quantity of Urine.*—An increase of the quantity of urine does not occur in stomach diseases, but there may be a decrease, and this begins as soon as the motor power of the stomach is materially restricted. In the amount of urine excreted in twenty-four hours we have a good measure of the magnitude of this disturbance. If, for instance, the amount of urine in twenty-four hours reaches only 300 to 500 grammes, when the liquid is taken up freely, we can assume a severe degree of motor disturbance; when there is a diuresis of 500 to 900 grammes, a disturbance of medium degree, and over 900 grammes, a slight one. I observed normal amount of urine twice in carcinomatous stenosis of the pylorus in spite of a high degree of motor insufficiency. In both cases diabetes had formerly been present, so that actually a polyuria had remained. Signs of contraction of the kidney were not present.

2. *The Reaction.*—Already under normal conditions, as Bence Jones[1] first discovered in the year 1819, there is an alteration of the reaction of the urine of such a nature that after the meal it becomes at first less acid, then neutral, and finally (three hours after breakfast, five to six hours after a full dinner) alkaline. Gradually the urine becomes acid again. Bence Jones himself already ascribed these variations to acid excretion from the blood and acid production in the stomach. These investigations, con-

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[1] Bence Jones, Philosophic Transactions, 1819, P. 235.

firmed by Robert,[2] Owen Rees, Quincke,[3] Maly,[4] and Stein[5] were then broadened by Görges.[6] Görges came to the following results: the urine suffers a continual loss of acid after each meal of mixed diet, so that it reacts alkaline after two hours, in three to five hours the alkalescence increases and then again it turns acid. With purely animal diet the loss of acid is less than with mixed diet, and with strictly vegetable diet loss of acid is likewise to be observed, but the urine does not turn alkaline.

The amount of acid is greatest in the morning urine. The acid reaction of the urine is increased by the introduction of HCl into the stomach, and the alkalescence of the urine can be thus prevented. A contrary action follows the ingestion of the alkaline carbonates.

Quincke[7] first pointed to the alkalescence of the urine as caused by the loss of acid in the stomach through vomiting, lavage, etc. When the gastric contents do not contain secreted hydrochloric acid, but only organic acids, the urine naturally remains acid, so that the detection of the reaction of the urine after lavage of the stomach or vomiting may, under certain circumstances, serve as diagnostic and prognostic indices.

Sticker and Hübner[8] have tried to establish the relations between secretion of the gastric juice and the reaction of the urine. As a valuable and interesting result of the experiments of these investigators must be emphasised the fact that *the decrease of acid in the urine does not occur* (or that on the contrary, the acid reaction is enhanced) *when, with the ingestion of food into the stomach, no appreciable collection of hydrochloric acid goes on in the stomach.* Ringstedt[9] tested and confirmed the statements of Sticker and Hübner by exhaustive studies of the acidity of the urine under different experimental conditions.

From the foregoing investigations of the urine the following conclusions for the diagnosis of stomach diseases could be derived:—

(*a*) Alkalescence of the urine after washing-out of the stomach or vomiting speaks for hyperchlorhydria or gastrochylorrhœa. No change in reaction speaks for the formation of organic acids.

(*b*) Non-occurrence of the physiologic variations of the reaction of the urine, or increase of the acidity of the urine some time after the ingestion of the principal meal, speaks for deficiency or absence of HCl secretion in the stomach.

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[2] Robert, A Practical Treatise on Urinary and Renal Diseases, second edition, 1872, P. 48.

[3] Quincke, Correspondenzblatt für Schweizer Aerzte, 1874.

[4] Maly, Liebig's Annalen, 1874, Bd. 173, P. 227.

[5] Stein, Deutsches Archiv für klin. Medicin, 1876, Bd. 18, P. 207.

[6] Görges, Archiv für experiment. Pathol., 1879, Bd. 11, P. 156.

[7] Quincke, *l.c.*, and Zeitschrift für klin. Med., 1884, Bd. 7, Supplementheft, P. 25.

[8] Sticker and Hübner, Zeitschrift für klin. Medicin, 1887, Bd. 12, P. 114.

[9] O. T. Ringstedt, Studier öfver aciditäten i menniskans urin under fysiologiska och patologiska förhallanden. Hygiea, Bd. 15; reference in Maly's Jahresb. f. Thierch., Bd. 20, P. 196.

E. Friedberger[10] has studied the behaviour of the acidity of the urine in exclusive rectal feeding, and found that when the food is given thus an increased excretion of acid appears in the urine. While diminution of the acidity in the urine after the ordinary feeding is to be considered as a measure for computing the acidity of the stomach, the increase of the acidity in the urine after rectal feeding yields a measure for the intestinal digestion.

Under pathologic conditions alkaline urine occurs in severe motor disturbances of the stomach, especially in benign stenosis of the pylorus.

3. *The Specific Gravity.*—This may naturally be raised under certain conditions, as in scanty diuresis, as most commonly observed in dilatation of the stomach. Otherwise the specific gravity has no diagnostic significance.

4. *The Chlorides.*—Jaworski and Gluzinsky[11] first demonstrated that in superacidity the chlorides present in the urine are extraordinarily decreased. Furthermore, M. Rosenthal[12] observed considerable diminution of chlorides frequently associated with cardialgia and vomitus, in forms of superacidity due to mental overexertion, strong emotion, or migraine.

Especially was this so in cases with long-continued or obstinate vomiting, where even slight amounts of food were scarcely tolerated by the stomach. Also Sticker[13] and likewise Gluzinsky[14] have given their attention to this question, and agree in the fact that there is a diminution of the chlorides in the urine when there is a supersecretion of HCl. Moreover, the diminution of chlorides in the urine may be the sequel of diminished absorption from the gastro-enteric tract (carcinomatous stenosis of the pylorus). Stroh,[15] as a result of his investigations upon the elimination of chlorine in stomach diseases, came to the conclusion that the same is entirely normal in ulcus ventriculi without complications, and in nervous dyspepsia and superacidity, also in chlorosis, etc. Increase of the chlorine elimination was never found, but, *on the contrary, decrease is regularly found in chronic supersecretion and ectasia of the stomach.* Robin[16] likewise found noticeable diminution of the excretion of the chlorine in hypochlorhydria, although it may occasionally be increased. Bouveret[17] takes the view that the elimination of chlorine in supersecretion depends on various circumstances, for example, the kind of food or the occurrence of vomiting, on account of which the chloride elimination can possess no proper significance. On the other hand, in cases of supersecretion the figure which represents the proportion of chlorides to urea is said to be greater than under normal circumstances. In cases with deficiency of HCl, however, as, for example, in cases of carcinoma, the proportion of the chlorides to urea is smaller than under normal circumstances. A considerable diminution of the value of chlorine has been observed in patients with carcinoma. Thus Fr. Müller[18] found diminution of the chlorides in all cases of carcinoma that he had an opportunity to investigate. This diminution of the chlorine excretion is, however, no specific sign of carcinoma, according to the investigations of v. Noorden and Gärtig,[19] as well as of Laudenheimer;[20] when retention of chlorides occurs in the body it depends on other circumstances, especially on retention of water (œdema, ascites, etc.).

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[10] E. Friedberger, Deutsches Archiv für klinische Medicin, 1899, Bd. 65, Heft 5 and 6.

[11] Jaworski and Gluzinsky, Sitzungsprotocoll der poln. Naturforscher und Aerzte vom 2 June, 1889; cited in Riegel, Magenkrankheiten, Th. 1, P. 46.

[12] Rosenthal, Berliner klin. Wochenschrift, 1887, No. 28.

[13] G. Sticker, Berliner klin. Wochenschrift, 1887, No. 41.

[14] Gluzinsky, *ibid.*, No. 52.

[15] Stroh, Inaug.-Diss., Giessen, 1888.

[16] Robin, in G. Lyon, L'analyse du suc gastrique, P. 89.

5. *The Phosphates.*—According to Gubler and Robin,[21] the amount of phosphates (normally 1.8 to 3 grammes) is very much increased in hypochlorhydria, in a case of Gubler's up to 12 grammes in the twenty-four hours' urine. According to Robin, the increase in the phosphates is more constant than the diminution of the chlorides.

Further, Fr. Müller[22] found in cases of carcinoma increase of the phosphates in those cases in which the food ingested was small, and it seemed independent from the process itself.

6. *The Sulphuric Acids.*—As is known, we distinguish between the sulphuric acid derived from the albuminous bodies—which we know as preformed sulphuric acid—and the conjugate sulphuric acids (ether-sulphuric acid) which are derived from the combination of sulphuric acid with the products developed by intestinal putrefaction. The total amount of the preformed and the ether-sulphuric acids reaches 1.3 to 3.1 grammes a day (average, 2.0 to 2.5 grammes), of which, according to van den Velden, the ether-sulphuric acids amount to about 0.09 to 0.6 gramme (average, 0.27 gramme), so that the relation of the conjugate to the preformed sulphuric acids may be considered about 1 : 10. According to Baumann and Herter, however, this relation varies widely, so that it may fluctuate between 4.2 and 27.0.

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[17] Bouveret, Rev. de méd., 1891, No. 7.

[18] Fr. Müller, Zeitschrift für klin. Medicin, 1889, Bd. 16, P. 496.

[19] Gärtig, Inaug.-Diss., Berlin, 1890.

[20] Laudenheimer, Zeitschrift für klin. Medicin, 1892, Bd. 21, P. 573.

[21] Robin, *l.c.*, P. 89.

[22] Fr. Müller, Zeitschrift für klinische Medicin, 1889, Bd. 16, P. 537.

Of pathologic significance solely is an excessive excretion of the *ether-sulphuric* acids (F. Müller,[23] Kast,[24] Salkowski,[25] v. Noorden[26]). According to the latter, 0.25 gramme of conjugate sulphuric acid may serve as an average limit in healthy persons.

Kast[27] and Wasbutski[28] found, independently of each other, that in the absence of hydrochloric acid the conjugate sulphuric acids increase considerably. Biernacki[29] arrived at the same result in experiments on human beings, and Ziemke[30] by experimenting on dogs, the gastric juice of which had lost its usual action by the administration of meat deprived of chlorine. v. Noorden,[31] on the contrary, showed that in simple inacidity without other complications the ether-sulphuric acids lie within the limits of the normal condition, and I[32] found the same in a case of stenosis of the duodenum with permanent absence of hydrochloric acid.

G. Hoppe-Seyler[33] also tells of the fact that in gastric diseases, when fermentative products accumulate in great quantities, there is not always an increased excretion of ether-sulphuric acids. On the other hand, as the excellent experiments of B. Mester[34] have shown, there may be an increase of the ethereal sulphates in the urine as soon as putrid substances are put into the stomach, free of acid. On the contrary, according to Mester's investigations, there is no increase in ethereal sulphates when the stomach contains hydrochloric acid.

On the whole, however, at the present time, the excretion of the ether-sulphuric acids cannot be regarded any longer as a measure for estimating gastro-enteric putrefaction, as they constitute only a part of the total aromatic combination.

7. *The condition of nitrogen* in the urine in stomach diseases is not without significance in a diagnostic, prognostic, and therapeutic respect. As regards diagnosis, there are the former investigations of Rommelaire[35]

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[23] Fr. Müller, Zeitschrift für klinische Medicin, 1887, Bd. 12, P. 63.

[24] Kast and Baas, Münchener medicinische Wochenschrift, 188, P. 55.

[25] Salkowski, Zeitschrift für physiologische Chemie, 1888, Bd. 12, P. 223.

[26] v. Noorden, Zeitschrift für klin. Medicin, 1890, Bd. 17, P. 528 and 529.

[27] Kast, Festschrift zur Eröffnung des allgemeinen Krankenhauses zu Hamburg-Eppendorff, 1889.

[28] Wasbutski, Archiv f. experim. Pathol. u. Pharmak, 1890, Bd. 26, P. 133.

[29] Biernacki, Centralblatt für die med. Wissenschaft, 1890, No. 49 and 50.

[30] E. Ziemke, Inaug.-Diss., Halle, 1893.

[31] v. Noorden, *l.c.*

[32] Boas, Deutsche medicinische Wochenschrift, 1891, No. 28.

[33] G. Hoppe-Seyler, Zeitschr. f. physiol. Chemie, 1888, Bd. 12, P. 1-32.

[34] B. Mester, Zeitschrift f. klin. Medicin, 1894, Bd. 24, Heft 5 and 6, P. 440.

[35] Rommelaire, Journal de méd., de chir. et de pharmac. de Bruxelles, 1883, 1884, 1885, 1886.

and later those of Thiriar,[36] Kirmisson,[37] and many others, which showed that in patients afflicted with carcinoma in general and those with carcinoma of the stomach in particular, the amount of N is considerably reduced. This behaviour was said to be of significance diagnostically, as distinguishing carcinoma of the stomach from other benign processes. Dujardin-Beaumetz[38] also found the amount of N reduced to 7 to 14 grammes (normally, 30 to 40 grammes), while in ulcerative gastritis it was 20 to 26 grammes, and in ulcus ventriculi 20 grammes. The credit of having solved this question belongs without doubt to Fr. Müller[39] and G. Klemperer,[40] who were the first to pursue its study rigidly and assiduously. They found an increased excretion of N characteristic of carcinoma, which in many cases is independent of the ingestion of food and is indicative of a waste of the tissue albumen. Even a generous diet may in this case not attain to the level of N equilibrium.

In cases other than carcinoma, Kolisch[41] found in a patient with severe gastric hæmorrhages an amount of nitrogen of 19 to 20 grammes, which is thrice the ordinary hunger-value. Kolisch attributes the large amount of N elimination to a pathologic decomposition of albumen. Neusser[42] also found 40 grammes of N in the urine in a case of ulcer.

8. *Peptic Ferments.*—Brücke[43] first called attention to a pepsine-like body in the urine. This observation was confirmed by a great number of other investigators (Grützner, Sahli, Leo, Gehrig, Stadelmann, Patella). Trypsin also, according to the investigations of Sahli and Gehrig, is said to be found in the urine, which statement, however, has been refuted by Leo, Stadelmann, and Grützner. Grützner[44] does not go so far as to deny entirely the occurrence of trypsin. He believes that in examinations of small amounts of fresh urine trypsin may be found in traces, and that in various affections of the pancreas which may cause retention of the pancreatic juice, larger amounts of trypsin may be demonstrated. Further, lab-ferment has

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[36] Thiriar, Congrès français de chirurgie, 1885; Semaine médicale de Paris, 1885, No. 17.

[37] Kirmisson, *ibid.*

[38] Dujardin-Beaumetz, Gazette des hôpitaux, 1884, P. 175; Gazette hebdomadaire, 1884, No. 31.

[39] Fr. Müller, Zeitschrift für klinische Medicin, 1889, Bd. 16, P. 496; there, also, other literature.

[40] G. Klemperer, Berliner klinische Wochenschrift, 1889, No. 40.

[41] Kolisch, Wiener klinische Wochenschrift, 1897, No. 26.

[42] Neusser, quoted by F. Blumenthal, Pathologie des Harns am Krankenbett, 1903, P 410.

[43] Brücke, Sitzungsberichte der kais. Akad. d. W., 1881, Bd. 44, P. 618.

[44] P. Grützner, Deutsche medicinische Wochenschrift, 1891, No. 1.

been found in the urine, first by Holovtschiner[45] and then by Hoffmann[46] and by myself.[47] Furthermore, Grützner, Gehrig, Holovtschiner (*l.c.*), Breusing,[48] v. Jaksch,[49] Leo,[50] and B. Rosenberg[51] have found also a ferment with a diastatic action in the urine.

The diagnostic significance of pepsine seems to be very slight. According to the investigations of Stadelmann,[52] pepsine is not absent even in the severest febrile cases, but, on the contrary, is even increased; likewise in diabetes. Also Leo, who originally was inclined to assign a diagnostic rôle to the absence of pepsine, came on the basis of further experiments, to the conclusion that diminution of pepsine occurs also otherwise without any special cause.

Likewise experiments (not published) made in my laboratory by Edgar Gans have shown that there is no regularity in the excretion of pepsine, which would be of any diagnostic value. Thus, he found in severe cases of catarrh of the stomach, in which the contents showed neither pepsine nor pepsinogen, peptic capability of the urine, while in other cases it sometimes failed to appear when the secretion of the gastric mucous membrane was well preserved or increased.

Bendersky[53] also came to the very same results, and Brunner[54] alone is of the opinion, based on post-mortem examination, that pepsine is supposed to be always absent in gastric cancer; likewise, according to E. Friedberger,[55] the deficiency of pepsine in the urine in cases of diminished secretion of juice is so decidedly pronounced that it may be regarded as of diagnostic significance.

According to my examinations there is neither any diagnostic significance to be attached to the *lab-ferment,* as it also may be present or absent in pathologic cases independently of the condition of the secretion of the gastric juice. Also many variations and quantitative discrepancies as to the amount of ferment occur in one and the same individual, as I have frequently observed.

No investigations have as yet been made concerning the possible diagnostic significance of the *diastatic ferment*. Leo (*l.c.*) could not establish a constant decrease of the same under pathologic conditions. In a number of diabetics, however, there was shown marked increase of the diastatic ferment in the urine.

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[45] Holovtschiner, Virchow's Archiv, 1886, Bd. 104.

[46] Hoffmann, Pflüger's Archiv, 1887, Bd. 41, P. 148.

[47] Boas, Zeitschrift für klinische Medicin, 1888, Bd. 14, P. 264.

[48] Breusing, Virchow's Archiv, 1887, Bd. 107, P. 186.

[49] v. Jaksch, Klinische Diagnostik innerer Krankheiten, 1901, 5. Auflage. See there the rest of the literature.

[50] Leo, Verhandlungen des Kongresses für innere Medicin, 1888.

[51] Rosenberg, Inaug.-Diss., Tübingen, 1890.

[52] Stadelmann, Zeitschrift für Biologie, 1887, Bd. 24; 1888, Bd. 25.

[53] J. Bendersky, Virchow's Archiv, 1890, Bd. 121, Heft 3.

[54] W. Brunner, Gaz. lekarska, 1890, No. 21.

[55] E. Friedberger, *l.c.*

Bendersky likewise noted the same phenomenon in two diabetics, and he mentions that also in diabetes insipidus he found occasionally more diastatic ferment than under normal conditions.

9. *Abnormal urinary ingredients* are very common in chronic affections of the stomach, especially those of a grave nature. Among them are *albumen, peptone (albumose), acetone-like substances, indigo derivatives,* and *volatile fatty acids.*

(*a*) *Albuminuria,* apart from complications of kidney and stomach diseases, occurs in gastric diseases, partly transitorily, and partly constantly to a slighter extent. According to v. Noorden[56] albuminuria is found to a somewhat larger degree after the occurrence of spasms of the stomach, and especially after severe gastric hæmorrhages. Fr. Müller calls attention to the frequency of albuminuria in carcinoma of the stomach, occurring, as it does, although perhaps only transitorily, in 35 to 72 per cent. of all cases of carcinoma of the different organs.

According to my observations concerning cancers of the œsophagus, stomach, intestine, and liver, more than traces of albumen were decidedly the exception.

(*b*) *Albumosuria** is found in a great number of acute and chronic diseases, and, according to Fischel, it is a regular occurrence in the puerperium. We may distinguish a hæmatogenic albumosuria (scurvy, v. Jaksch), a pyogenic (suppurative processes in the body, Hofmeister, Maixner, v. Jaksch), enterogenic (Maixner, Pacanowski), and hepatogenic (Pacanowski, Alison), and finally a necrogenic, that was observed by A. Köttnitz, in the puerperium, following the death and maceration of the fœtus.

The enterogenic albumosuria, first described by Maixner,[57] then by Pacanowski,[58] and of late years by Robitschek[59] and Chvostek and Stromayr,[60] is of special interest for us. The first-named author found a regular albumosuria in ulcerative processes of the gastro-enteric tract, which he traced to the direct absorption of albumoses through the decomposing tumour (and not in the normal way). Pacanowski, who, on the whole, could substantiate the statements of Maixner, considers the destruction of the tissue as the cause of the albumosuria, which view appears to be correct.

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* Here we do not mean genuine peptone, as stipulated by Kühne, but peptone in its former sense; recently, therefore, peptonuria has been more correctly designated as albumosuria.

[56] v. Noorden, Lehrbuch der Pathologie des Stoffwechsels, Berlin, 1893.

[57] Maixner, Zeitschrift für klinische Medicin, 1884, Bd. 8, P. 534.

[58] Pacanowski, Zeitschrift für klinische Medicin, 1885, Bd. 9, P. 428.

[59] Robitschek, Zeitschrift für klinische Medicin, 1894, Bd. 24, Heft 5 and 6, P. 556.

[60] Chvostek and Stromayr, Wiener klin. Wochenschrift, 1896, N. 47.

This author also found albumosuria in severe diseases of the liver (carcinoma, acute atrophy, phosphorus poisoning). Robitschek found albumosuria only twice in seven cases of carcinoma of the stomach, and also in one case of carcinoma of the rectum and in one case of ulcus ventriculi. v. Aldor[61] found albumosuria in four cases of carcinoma of the stomach and in one case of cancer of the peritoneum. Alison[62] has also reported the occurrence of albumoses in the urine after cholelithiasis. Chvostek and Stromayr, finally, found alimentary albumosuria in cases of tuberculosis of the intestine with ulcerative destruction of the intestinal mucous membrane. Reports of frequent occurrence of albumosuria, which are often found in the literature, must be accepted cautiously,* as the precautions necessary for the demonstration of albumoses (especially mistaking it for urobilin) are not always observed. v. Aldor is inclined to regard albumosuria in carcinoma as an anomaly of metabolism. At the present time, a diagnostic significance cannot be assigned to albumosuria.

(*c*) *Acetone and Diacetic Acid in the Urine.*—Already Petters and Kaulich called attention to the connection between gastric disturbances and acetonuria. This observation has been placed on a firm foundation through the demonstration of acetone in the gastric contents and the fæces, as shown by v. Jaksch.[63] Litten[64] was the first to describe a characteristic dyspeptic symptom-complex (coma dyspepticum), distinguished, after prodromal symptoms of several days' duration, by acute gastric disturbances (nausea, eructation, flatulency, anorexia, vomiting, constipation or diarrhœa, and by characteristic nervous troubles (frontal headache, sleeplessness, depression, restlessness, etc.). At the same time we may observe a peculiar aromatic odour of the exhaled air, with the appearance of the well-known Burgundy-red colouration of the urine with iron chloride (diacetic acid). In a series of very careful investigations made especially in regard to the occurrence of acetonuria in digestive disturbances, Lorenz[65] found acetonuria and diaceturia in a great variety of primary and secondary, acute and chronic gastric affections. The acute cases of gastro-enteric catarrh after the ingestion of tainted meat were characterised by the *constant* presence of acetone in the urine, while diacetic acid occurred in varied amounts, from complete absence to the very pronounced presence. Acetonuria does not occur constantly in chronic gastro-enteric affections, but runs parallel with the variations and especially with the intensity of the symptoms of the disease.

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* That albumosuria in carcinoma of the stomach is not a common finding, is to be inferred from the fact that, for example, B. Schultes found, among nine cases, only one of albumosuria, and in this case there was continuous fever.

[61] v. Aldor, Berliner klinische Wochenschrift, 1899, No. 35 and 36.

[62] Alison, Arch. générales de médecine, 1887-88.

[63] v. Jaksch, Zeitschrift für klinische Medicin, 1884, Bd. 8, P. 36.

[64] Litten, Zeitschrift für klinische Medicin, 1884, Bd. 7, Supplemenatb, P. 81.

[65] Lorenz, Zeitschrift für klinische Medicin, 1891, Bd. 19, P. 79.

In severe cases of gastro-duodenal catarrh, as well as in cases of acute gastro-enteritis, and likewise also in intestinal occlusion, as, for example, in the wake of coprostasis, Lorenz could demonstrate acetone and diacetic acid in the urine, and in the cases last named also in the fæces. This author found acetonuria also in cases of tapeworm, in acute peritonitis, in gastric crises, in the periodic vomiting of v. Leyden, in cholelithiasis, in lead colics, in hysteria with gastro-enteric symptoms, etc.

My own experience on this question has been confined almost exclusively to gastro-enteric affections. I found acetone or diacetic acid now and again in severe forms of gastrectasis (in one case with exquisite odour of acetone in the exhaled air), further in two cases of stenosis of the duodenum, and repeatedly in carcinoma of the stomach, although only in the last stages of the same. F. Blumenthal[66] likewise found acetone as a sign of inanition and intoxication in carcinoma and ulcus ventriculi. Furthermore, Blumenthal observed cases of ulcus ventriculi in which acetone appeared when the ingestion of food was markedly reduced, but disappeared as soon as abundant nutritive enemata were administered.

Diagnostic conclusions cannot be drawn from the presence of acetone in the urine, according to the investigations to date, except that a *constant* occurrence of acetone points to an increased decomposition of albumen in the alimentary canal.

(*d*) *Indican and Indigo-red in the Urine.*—The presence of indican in the urine possesses a secondary significance for the diseases of the intestinal tract, especially since we know that also in harmless processes in the intestinal tract, for example, coprostasis, indican may appear in the urine. Häberlin[67] found in 20 per cent. of cases of gastric carcinoma no increase of indican, in 60 per cent. a moderate, and in 20 per cent. a very marked increase.

Indigo-red (indigrubin) is, according to the very thorough investigations of Rosin,[68] a pigment of the urine related to indigo-blue, and is to be considered, according to Rosenbach,[69] who first pointed out its appearance and determined its pathologic significance, as an expression of serious metabolic disturbances and is of diagnostic value. According to Rosenbach, the reaction is especially present:—(1) in severe affections of the intestines that lead to insufficiency of the intestines, therefore without exception in occlusion of the intestines; (2) in severe diarrhœa; (3) in patients with chronic affections attended with severe disturbances of nutrition.

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[66] F. Blumenthal, Pathologie des Harnes am Krankenbett, 1903, P. 171.

[67] Häberlin, Deutsches Archiv für klinische Medicin, 1889, Bd. 45, Heft 3 and 4, P. 339.

[68] Rosin, Centralblatt für klin. Medicin, 1889, No. 29.

Its persistent appearance is said to serve as a *signum mali ominis.* Investigations by Ewald,[70] however, have materially limited its diagnostic and prognostic value. This investigator saw a negative reaction in cases of severe intestinal affections (for example, carcinoma of the colon) and also in phthisis with grave nutritive disturbances, where it was plainly absent (first degree, Ewald). According to Ewald, the pigment, like indican, is an expression of an abnormal intestinal metabolism, and that of processes of decomposition in the small intestine. As to prognosis, the appearance and the persistent presence of the pigment is certainly unfavourable; but the disappearance does not precede a favourable turn, but follows it. P. Abraham[71] and others have arrived at similar results. Biermer[72] furthermore saw the reaction not only in severe diseases, but also in slight affections of the stomach and intestine.

(*e*) *Volatile Fatty Acids.*—F. Rosenfeld[73] recently investigated the relation of the volatile fatty acids of the urine to gastric diseases, and found them increased in ulcer with superacidity or normal acidity, but diminished in conditions of stagnation in the wake of atony, ectasia, and gastroptosis, as far as they are associated with inacidity or subacidity; often increased in carcinoma of the stomach, when inacidity or subacidity is present. Rosenfeld considers these findings important for diagnosis.

In the foregoing only the abnormalities of the urine important for the symptomatology of diseases of the digestive apparatus have been collected; regarding the tests for these, as well as for rare anomalies, we have to refer to the text-books on clinical diagnosis and on urine.

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[69] Rosenbach, Berliner klin. Wochenschrift, 1889, No. 1 and No. 22-23.

[70] C. A. Ewald, Berliner klinische Wochenschrift, 1889, No. 44.

[71] P. Abraham, Berliner klinische Wochenschrift, 1890, No. 17.

[72] Biermer, Berliner klinische Wochenschrift, 1889, P. 968.

[73] F. Rosenfeld, Verhandl. des Vereins für innere Medicin, Januar 5, 1903.

CHAPTER VIII.

Diagnostic Significance of the Examination of the Blood in Gastric Diseases.

Certain authors have endeavoured to apply the methods lately available for the examination of the blood to the diagnosis of stomach diseases. In the first place is to be considered the differential diagnosis between the most common malignant affection of the stomach—carcinoma—and the benign processes of the gastric mucous membrane. Opinions as to the value of these methods are still divided; as an average judgment, however, we may take the ground that as yet the blood-examination does not afford any decisive means for the differential diagnosis. In this we follow essentially the very thorough and soundly critical monograph of E. Reinert,[1] but take also into account the other contributions referring to this subject.

F. Müller,[2] Oppenheimer,[3] and Häberlin[4] state that the coloured elements are not, as a rule, diminished in ulcer, but are appreciably diminished in carcinoma. On the other hand, Leichtenstern[5] found a diminished amount of hæmoglobin in three cases of ulcer, while Oppenheimer[6] could find no diminution of the same in twelve cases. Reinert,[7] on the other hand, in two cases found the number of red blood-corpuscles, as well as especially the amount of hæmoglobin, markedly diminished. Osterspey[8] found in nine cases of ulcer, some with and some without hæmatemesis, regular diminution of the number of red blood-corpuscles and of the amount of hæmoglobin, and in some a decrease in the white elements. From this it follows, as Osterspey correctly remarks, that *neither the normal nor abnormal blood-texture is characteristic of ulcus ventriculi.*

The investigation of the blood in *carcinoma of the stomach* has likewise furnished no unequivocal results. It is true that a decrease in the red corpuscles, along with a diminution in the amount of hæmoglobin, has been observed in most cases (Leichtenstern, Laache, Malassez, Schneider, Daland and Radler, Häberlin, Osterspey, Mouisset and Tolot, and others), but this alteration of the blood, quite apart from the exceptions, is not at all characteristic of carcinoma of the stomach. Also the blood-findings do not agree so far as the leucocytes are concerned; besides reports as to increase of the white blood-corpuscles (Leichtenstern, Sörensen, Potain) we meet with others with a normal condition (Lépine, Laache, Schneider, Osterspey, and others).

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[1] E. Reinert, Die Zählung der rothen Blutkörperchen und deren Bedeutung für Diagnose und Therapie, Leipzig, 1891.

[2] Fr. Müller, Verhandlungen des Vereins für innere Medicin, Jahrgang 7, 1888, P. 378.

[3] Oppenheimer, Deutsche medicinische Wochenschrift, 1889, No. 42-44.

[4] Häberlin, Münchener medicinische Wochenschrift, 1888, No. 22.

[5] Leichtenstern, Untersuchungen über den Hämoglobingehalt im gesunden and kranken Zustande, Leipzig, 1878.

[6] Oppenheimer, *l.c.*

[7] Reinert, *l.c.*

[8] Osterspey, Berliner klinische Wochenschrift, 1892, No. 12 and 13.

From this follows the unlikelihood of distinguishing in doubtful cases, by the blood-examination, between ulcer and carcinoma, or benign and malignant stenosis of the pylorus, as Häberlin has stated, and that the less so because in doubtful carcinoma we are not dealing with pronounced cachexia, on account of which the blood-findings may show either no clear deviations from the normal or what is merely significant.

Besides these changes in the blood, v. Jaksch,[9] as well as Grawitz[10] and Strauer,[11] have studied the amount of albumen in the blood in cases of carcinoma. According to the investigations of the last-named authors, who studied all the factors in consideration (number of red and white blood-corpuscles, dry residue of the blood, dry residue of the serum, specific gravity), the blood in advanced cachexia appeared subnormal in all the named conditions.

The determination of the specific gravity of the blood may have a certain corroborative significance (Hammerschlag, R. Schmaltz). According to Schmaltz this reaches, on the average, 1.0591; according to Peiper, 1.0550; in women, according to Schmaltz, 1.0562, according to Peiper, 1.0535. In cases of anæmia due to various causes, the specific gravity is appreciably diminished. Of special interest is the finding of Peiper, namely, that in carcinoma and in one case of chronic gastritis the specific gravity was considerably diminished. According to observations of my own in four cases of gastric cancer, there was in two cases considerable diminution of the specific gravity (1.0272 and 1.0275), and in the other cases, advanced less, it was likewise diminished though to a slighter degree; in other benign affections of the stomach the specific gravity remained within the normal limits. In accordance with Schmaltz, I found in the majority of cases, although not regularly, along with the diminution of the specific gravity, a decrease of the amount of hæmoglobin. Devoto, also, using another method, found diminution of the specific gravity in a case of carcinoma, and in another case it was normal.

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[9] v. Jaksch, Zeitschrift für klinische Medicin, 1893, Bd. 23, Heft 3 and 4.

[10] E. Grawitz, Deutsche medicinische Wochenschrift, 1893, No. 51.

[11] Strauer, Zeitschrift für klinische Medicin, 1894, Bd. 24, Heft 3 and 4.

Recently Schneyer[12] called attention to an important differential sign between carcinoma of the stomach and benign processes. It consists in the fact that the presence of digestion leucocytosis is supposed to be evidence against carcinoma, but that a want of leucocytosis is not of any significance. Controlling tests of Hassmann, Hofmann, Sailer and Taylor, Chadbourne, Dolmatow, Rencki, Marchetti,[13] Krokiewicz,[14] Mouisset and Tolot,[15] and others show that the exceptions to the rule established by Schneyer are so numerous, that a diagnostic significance cannot be assigned to the digestion leucocytosis.

Only scanty examinations have been made for the remaining gastric affections. Thus Leichtenstern found normal figures for hæmoglobin in gastrectasis and that in spite of significant loss of weight and appreciable marasmus, and attributes this to the diminished amount of water in the blood due to poor absorption. In two cases of Reinert's, the number of red blood-corpuscles was normal and the amount of hæmoglobin somewhat diminished. In one case under my own observation, likewise with noticeable marasmus, the amount of hæmoglobin (estimated according to Gowers) was 100 per cent. Osterspey found always normal counts for red and white blood-corpuscles in cases of anachlorhydria, gastritis chronica, and nervous dyspepsia, but in several cases he found noticeable diminution of the amount of hæmoglobin.

Pernicious anæmia establishes a very common blood alteration in severe gastric affections, and is found especially in connection with atrophy of the mucous membrane of the stomach. Pernicious anæmia is, however, no constant accompaniment of atrophy of the mucosa; and, on the other hand, deeply-seated alterations of the mucosa (Immermann, Quincke, and others) may be absent in pernicious anæmia. Besides accompanying atrophy of the mucous membrane, pernicious anæmia is found in connection with carcinoma of the intestinal tract as well as with ulcus ventriculi, associated with repeated though latent hæmorrhages. Diagnostic significance, therefore, cannot be attributed to pernicious anæmia, although when the latter is associated with severe gastric symptoms, and when grave disturbances of secretion (absence of hydrochloric acid and ferments) are existing, the diagnosis of atrophy of the gastric mucous membrane is naturally reinforced. For the differential diagnosis between carcinoma and pernicious anæmia, there is, according to Henry,[16] the significant fact that in the former the number of erythrocytes never falls below 1,500,000 in spite of considerable cachexia, while the fatal cases of pernicious anæmia always show an erythrocyte count under 1,000,000. Also, according to Krokiewicz,[17] the number of erythrocytes in carcinoma seldom falls below 2,000,000 per cubic millimetre.

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[12] Schneyer, Zeitschrift für klinische Medicin, 1895, Bd. 27, P. 475.

[13] Cited in Part II.

[14] Krokiewicz, Archiv für Verdauungskrankheiten, 1900, Bd. 4, Heft 1.

[15] Mouisset et Tolot, Revue de Médicine, 1902, P. 844.

The description of the methods of blood-examination does not lie within the limits of this work; the reader will find them in the special works on the pathology of the blood.

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[16] Henry, Archiv für Verdauungskrankheiten, Bd. 4, P. 1.

[17] Krokiewicz, *l.c.*

PART I

GENERAL DIVISION

II. GENERAL THERAPEUTICS

CHAPTER IX.

The Diet.

As to the appropriate feeding in diseases of the digestive apparatus we must take into consideration: 1. The question of necessary supply of food in general and in diseases of the digestive apparatus in particular. 2. The subjective complaints and observations of the patient (individual diet). 3. The results of the objective (physical and chemical) examination. All these points are of such great significance, that they must be considered separately.

A. The Amount of Food in General, and in Diseases of the Intestinal Tract in Particular.

In the feeding of a sick person the consideration of the general constitution, the advancement and improvement of his actual energy are the first and foremost goal. There are, however, diseases in which for a time, we must leave this out of consideration, and first look with care upon the organ which needs attention; after its healthy condition has been restored, we may again introduce into the body the full amount of the necessary nourishment. Sometimes, however, the nature of the illness makes our endeavour to increase the weight of the body and its strength illusory, as, for example, when a greater or lesser part of the ingested food is left unused, as occurs, for instance, in habitual vomiting, to whatever cause it may be due; or when the fat ingested is not absorbed, as in serious disturbances of the pancreas; or when the ingested foods do not take the ordinary paths of assimilation, but are dissolved in a manner contrary to the rule (toxogenic decomposition of albumen); or when, as in patients with fever, in spite of an increasing ingestion of nitrogen, there occurs a loss of albumen; or as in morbus Basedowii [Graves's disease] the intensity of the oxidation processes is greatly increased.

But even under the circumstances last mentioned the goal of compensating for the loss of nitrogen by an increase in the administration of albumen may be followed with success, as numerous examples have shown.

The principal question now to be answered is as to the amount of food ordinarily necessary.

Up to the present this question has been answered with accuracy only for the healthy person, and only to a slight degree for the sick, especially those suffering from digestive troubles. For the healthy those figures hold good which v. Voit published as an average measure for the sufficient feeding of adults and which have been used as the basis for all experiments in metabolism.

According to v. Voit, the daily requirements are:—

For men, 118 gm. albumen, 56 gm. fat, 500 gm. carbohydrates.
For women, 94 " " 45 " " 400 " "

It is to be remarked that these figures apply to a man of from 70 to 75 kilos of body weight, who works 10 hours a day at not too strenuous work, and that the amount of food for women is also estimated for a person at moderate work.

These figures, however, evidently represent the maximum of the food necessary for a normal person at work. Voit himself, but especially Pflüger, Bohland, Klemperer, Peschel, Kumagawa, Breisacher, Siven, Albu, and R. O. Neumann, [Chittenden, Mendel] were able to establish that the amount of albumen in the food may be greatly decreased. In this respect the experiments on himself by R. O. Neumann[1] are the most conclusive, extending, as they did, over a period of 746 days. They gave as an average amount of food necessary, of albumen, 74.2 grammes, of fat, 117 grammes, of carbohydrates, 213 grammes.

It follows from these figures that the decrease of the amount of albumen is, of course, possible, but only on a diet with an essential increase of the anitrogenous substances. These are therefore, in the proper sense of the word, albumen-sparers.

For the prevention of the loss of albumen, that is, for the accumulation of albumen, the carbohydrates prove themselves far more favourable than the fats, because a decrease of the combustion of albumen is always associated with an increase of the carbohydrates, whereas the administration of fats in increasing quantities does not necessarily decrease the consumption of albumen, but may even increase it.

We can also calculate the necessary amount of food of the normal individual in calories, as Rubner, especially in his classic investigation, has carried out. Upon these depends our present method of calculation in calories. Rubner founded the law, and established it experimentally, that the organic food-substances are capable of vicariously substituting one another according to the proportion of their caloric values (law of isodynamics).

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[1] R. O. Neumann, Archiv für Hygiene, 1902, Bd. 35, Heft 1.

The energy value of the food-substances is calculated (after Rubner) according to their physiologic caloric value, that is, the caloric value they yield on oxidation until only the end-products, the useless waste products, remain. As is known, the standard hereby employed is the calory, that is, the amount of heat necessary to raise the temperature of one kilogramme of water 1° C. Through experiments outside the body in the calorimeter, the caloric value of the individual food-elements up to the end-products, as which they leave the body, has been determined, and the average determined:—

1 gramme albumen = 4.1 calories
1 " carbohydrate = 4.1 "
1 " fat = 9.3 "
1 " alcohol = 7.0 "

It was further established by Rubner that the oxidation of food-elements in the body develops exactly as much heat as in the calorimeter, so that without any further examination we can estimate each food according to its caloric value and thus obtain a standard for the value of the nutrition in healthy and in diseased conditions.

The caloric values determined by Rubner (*gross calories*) are gross values, because in them the loss due to insufficient absorption of the food in the intestinal tract has not been considered. For the estimation of *clear calories* Rubner calculated a loss of 8 per cent., estimated from the average value of the calories of the fæces in mixed diet.

As an average quantity of food in twenty-four hours for healthy, adult, strong men of average height and 70 kilos weight, Rubner calculates:—

	Pure calories.	Calories per kilo.
At rest	2303	32.9
At slight manual work	2445	34.9
At medium manual work	2868	41.0
At hard manual work	3362	48.0

On subtraction of 8 per cent. from the 3055 gross calories in Voit's diet values, we get a very fair agreement with Rubner's calculation.

For the patient of about 70 kilos weight, working but little, we may consider (and it is easily remembered), a caloric value of about 2000 for the maintenance of weight, and of about 3000 for an improvement of the nutrition, as being satisfactory.

It is of great interest to determine the standard nutriments, through which we may attain the first-named amount of calories.

Two thousand calories are attained (according to Hirschfeld) by the oxidation in the organism of

Beef	1570 gm.	Butter	252 gm.
Veal	1360 gm.	Bread	848 gm.
Eggs	28 pieces.	Potatoes	2216 gm.
Fat cheese (cream)	524 gm.	Rice	580 gm.
Lean cheese (skimmed)	1120 gm.	Peas	610 gm.
Milk	**3460 c.c.**	Sugar	500 gm.
Cream	**1000 c.c.**		

In this table the caloric value of milk and cream interests us most particularly. We see from this how great a quantity of milk (about $3^{1}/_{2}$ litres) is necessary to sustain nutrition, but that we can get the same effect by the employment of a much less quantity of cream.

However important, doubtless, the calculation in calories for investigations of metabolism may be, we must object to the exaggeration that the whole nutrition of the sick has to be guided solely from the standpoint of the calculation of calories. We cannot think Ewald[2] entirely wrong, when, in sharply criticising these mistaken efforts, he holds the calculation in calories for all disturbances of the digestive organs to be entirely superfluous, as far as these are due to local affections. Ewald's dictum, however, applies only to the "laboratory" calorimetric estimation. The calculation in calories is not valueless in the hands of the thinking physician. Although they do not yield a mathematically rigid value, still in the individual case they give an approximate estimate of the oxidation value of the food. Thus, for example, the value of fat, especially of the milk-fat in cream, has been correctly appreciated for its nutritive qualities only since the recognition of its calorimetric energy.

We possess still another standard for the value of the introduced food, namely, the scales. "The scales," correctly observes v. Leyden, "are the surest measure for the progress of a dietetic cure." *Every physician who treats chronic diseases, yes, any physician at all, should, in my opinion, possess a precise weighing apparatus.*

Relative to the necessary nutriment in *chronic affections of the stomach* with which we will now concern ourselves, the investigations of v. Noorden[3] concerning the metabolism in chronic gastritis with loss of hydrochloric acid and superacidity are, above all others, of decisive significance. In conformation to the former observations of Czerny,[4] Ogata,[5] de Filippi,[6] and others on dogs whose stomachs had been removed, he arrived at the result,

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[2] Ewald, v. Leyden's Handbuch der Ernährungstherapie, 1898, Bd. 2, P. 247.

[3] v. Noorden, Zeitschr. für klin. Medicin, 1890, Bd. 17. Compare also Lehrbuch des Pathologie des Stoffwechsels, Berlin, 1893, P. 237.

[4] Czerny, Beiträge zur operativen Chirurgie, Stuttgart, 1878.

[5] Ogata, Archiv für Anatomie and Physiologie, 1883.

[6] de Filippi, Deutsche medicinische Wochenschrift, 1894, No. 40.

that in spite of absolute absence of hydrochloric acid-pepsine digestion the proteids are assimilated entirely and satisfactorily in the intestines. v. Noorden observed increase of nitrogen twelve times out of thirteen cases, on a diet that approached in general the usual diet. The length of the experiment lasted six to twenty-one days. The assimilation of fats and carbohydrates was also in no way diminished. Another proof for the vicarious action of intestinal digestion was furnished by A. Hofmann,[7] through investigations on a woman whose entire stomach was resected by Schlatter in the year 1897 on account of a diffuse carcinoma. In this case the assimilation of albumen as well as of fat was quite normal. It is true, there was not, as in the healthy, N equilibrium, for in six days of experimentation there was an addition of nitrogen of 0.1 to 1.14, on the whole of 4.25 grammes of nitrogen, which equals 26.5 albumen, corresponding to 725 grammes of muscle-meat. This increase of N was, however, not manifested as an increase in weight, so that the nitrogen retention must be considered as an accumulation of the circulating albumen. Strauss[8] came to similar results in his investigations of metabolism in cases of apepsia gastrica. These yielded the fact that the assimilation of the nitrogen and of the fat in the food was diminished only when there was coincident diarrhœa. In an experiment of metabolism instituted by Paul Mayer[9] in a case of apepsia gastrica with diarrhœa, only the elimination of nitrogen was appreciably impaired; that of the fat, however, was normal. Likewise, v. Noorden, in a case of superacidity, found the excrements made up in a way analogous to normal conditions.

From the mentioned investigations, v. Noorden concludes that marasmus in many chronic affections of the stomach (ulcus ventriculi, catarrh of the stomach, gastrectasis with supersecretion) is due to limited ingestion of food, and that in the treatment this fact would have to undergo radical changes. Without doubt, sub-nutrition in a great number of patients with gastric troubles (and this must be directly admitted) is one of the most important factors in their miserable condition, although this is not the invariable cause. To cite but an example, patients with benign and even with carcinomatous stenosis of the pylorus, sometimes have an excellent appetite, and the proper peptic power of the stomach is also well-preserved; but in spite of this, their ingestion of food is confined to very narrow limits, because every over-burdening of the stomach brings about the most distressing complaints (oppression, pains, eructations, nausea, and vomiting). As soon as the mechanical obstruction is relieved by operation, and the ingestion of food and its digestion are going on normally, an increase in weight immediately follows. On the other hand, the under-feeding may be the result of a persistent and insuperable loss of appetite (nervous anorexia, chlorosis, nervous dyspepsia, etc.).

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[7] A. Hofmann, Münchener medicinische Wochenschrift, 1898, No. 18.

[8] H. Strauss, Zeitschrift für klin. Medicin, 1900, Bd. 41, P. 280.

[9] Paul Mayer, Zeitschrift für diätetische und physikalische Therapie, 1901, Bd. 4, Heft 3.

Also of important significance are the valuable investigations of F. Müller[10] and G. Klemperer,[11] concerning the equilibrium of metabolism in *patients with cancer*, which agree fully in all essential points. They found that the amount of organic albumen is constantly diminished by the action of a particular poison, and it is not possible to maintain the normal weight by the most abundant possible feeding.

There are, however, exceptions to this rule, as v. Leyden[12] has quite correctly emphasised for a long time, and it is possible under favourable circumstances, by means of a sufficient method of feeding adapted to the characteristic conditions of the decomposition of the albumen, that the body may either maintain its N balance, or even increase it.

My own experiences in carcinoma of the stomach have been the following:—Increase of weight in patients with carcinoma of the stomach is exceptional, and is only attainable transitorially with the especially particular employment of highly concentrated foods. A pre-condition here also, however, is constant desire for food. As a rule, there is loss of weight or fluctuating additions in weight. The cause of it varies greatly; at one time it may be due to a mechanical condition (just as in benign dilatation) or again to insurmountable loss of appetite. The latter is not at all confined to mechanical obstruction, but occurs also in entirely unobstructed stomachs. Finally, there occurs, analogous to the views of Fr. Müller and Klemperer, a condition of progressive marasmus in spite of good appetite and normal motor function of the stomach. It is probably due to the fact that certain protoplasmic poisons are produced.

B. The Significance of the Subjective Complaints for the Diet.

In spite of the important elucidations furnished by the test of the functions of the stomach and the knowledge of the metabolism, the consideration of the subjective symptoms and the complaints possible in connection with the act of digestion are not to be disregarded in the plan of feeding. For the suitability of an article of food, apart from the objective digestibility, depends in so high a measure on the individual tolerance and other factors as yet unknown to us, that it is indispensible to regulate the quality and the quantity of the food with constant consideration of the individual observations.

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[10] F. Müller, Zeitschrift für klinische Medicin, 1889, Bd. 16, P. 496.

[11] G. Klemperer, Berliner klinische Wochenschrift, 1889, No. 40.

[12] E. v. Leyden, Deutsche medizinische Wochenschrift, 1890, No. 48.

If it would be still necessary to furnish a proof, we have it in *milk*. This article of food, par excellence in childhood, shows the most noteworthy individual variations as far as the gastro-enteric tract of adults is concerned. Some patients do not bear milk at all, not even with the employment of the customary corrigents; a second group may take only raw milk without having trouble; while others, again, as they allege, can take only boiled milk without uneasiness. In some patients milk causes a striking checking of peristalsis, for which no other cause can be found; in others, on the other hand, it causes diarrhœa. Here also the fact as to whether the milk is drunk raw or boiled, cold or warm, plays an undoubted part. Next to this the varying quality of the milk, which, regarded from the finely chemical standpoint, constitutes an extremely variable article of food, is certainly not the least cause for the varying tolerance. Further, objective disturbances occur in the digestive canal (chronic gastritis and gastroenteritis) which fully explain the unfavourable effects. But there are still other cases remaining in which every point of view is wanting for the comprehension of the bad effects of a seemingly so easily assimilable food. As with milk, patients act just as peculiarly in their reaction towards many other foods and drinks, so that a consideration of these variations is not to be disregarded.

On the other hand, we must use the statements of the patients concerning disturbances of digestion after certain foods only with the utmost critical judgment for our dietetic directions. The inclination is, indeed, to blame only too readily the substances usually regarded as difficult to digest, for this or that gastric disturbance. In order to estimate correctly the validity of such statements, we must only consider how extraordinarily complex such a simple meal as our customary breakfast is,* and how difficult it is even for an intelligent observer to make his mind clear as to what part of it is perhaps harmful. In order to determine the latter with certainty, I have recommended for years the following procedure, which I designate as a substitution method. For example, we wish to test as to whether the patient's morning cup of coffee is responsible, as alleged, for his indisposition. Then I advise the patient to take the customary breakfast for three days, with the same quantities of food, and same accessories, except that the coffee is to be omitted, but that the milk and sugar which may be the usual additions are to be taken. A careful observation may show now the influence of the coffee. Nevertheless, we cannot be certain even now, as we have given the coffee in infusion with water, and perhaps the latter element alone is responsible for the indisposition (for example, eructation). For greater certainty we should then advise the patient to take for one or two days a breakfast consisting of the remaining substances plus lukewarm water plus sugar, all in the same quantities. This way is, as I acknowledge, very complicated, and the difficulty increases considerably if a more generous meal is ingested, but it is *the only rational way to arrive at clearness concerning the causes of the subjective disturbances.**

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*[The customary breakfast in Germany is, as a rule, only a cup of coffee with milk and sugar, and a roll (with or without butter).]

The *appetite* plays an important rôle in the subjective symptoms, and, as the experiments of Pawlow already mentioned (page 24) have shown, also in the objective symptoms on the nature of which alone, as has already been emphasised, may depend sometimes the whole effect of nutrition. If we omit the rare cases of increased sensation of hunger, we have to do usually with a diminution of the desire for food, whether the latter be dependent on fear of pain (ulcus ventriculi, superacidity), or on an abnormal fulness of the stomach which prevents the sensation of hunger or causes easy satiation. Further, there exists a "nervous" anorexia, and it is the province of the most important requirements of a careful anamnesis to determine whether the loss of appetite is the sequel or the symptom of an organic disease of the stomach or of a general nervous depression, anxiety, worry, grief, and other abnormal sensations. Finally, it must be pointed out that a loss of appetite may also be artificial, that is, may be dependent on *perverted* dietetic measures (too monotonous a diet, prohibition of refreshments, of smoking, drinking wine or beer, etc.). It is evident that the regulation of the appetite in the first place has to be preceded by the removal of the causes inciting the disturbances of appetite. In as far as we succeed as to the removal of these causes, we can also improve the regulation of the appetite. In so far as this is not the case, we will not succeed, even by forced feeding, as recommended by v. Noorden, and even with the best intentions of physician and patient, in obtaining our aim, except very transitorily.

C. The Significance of the Objective Methods of Examination for the Dietetic Measures.

The objective examinations materially supplement the results of the subjective disturbances. They depend, on the one hand, on our knowledge of physiology and pathology in general, and on the other on empiric or experimental experiences in the healthy or sick, and finally on the results of investigation in the individual case. All these factors play a part in the regulations of the diet, and, as a matter of course, are principally the results of the physical and chemical investigations.

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* It is easier to substitute tea for coffee, and after a successful result to return again after a lapse of a few days to the coffee, while the quantities themselves remain the same.

Not only the disturbances of the gastro-enteric canal belong to these objective causes, but also (and that especially) those of *the oral cavity,* and here, again, *the condition of the teeth.*

The importance of sufficient mastication has been set forth anew through the labours of Schreuer and Alfred Riegel.[13] Through these it was shown that the deficiency of mastication induced a greater or lesser deficit of the amount of the hydrochloric acid, when mastication was concerned with the ingestion of carbohydrates. When albuminous foods are not properly masticated, the effect varies, according to whether the stomach contains a normal or an excessively high or a diminished amount of acid. Under the first-named circumstances the direct stimulating effects of these substances upon the hydrochloric acid secretion may fully compensate the inhibiting influence of the deficiency of mastication. In subacidity, however, the stomach is no longer capable of such a compensation. Here, indeed, the act of mastication is absolutely indispensable. From this follows the old rule that *the treatment of the patients suffering from diseases of the stomach must begin with the teeth.*

The admixture with saliva seems to have a somewhat slighter effect on the secretion of gastric juice. While Sticker and Biernacki regard the insalivation as an impulse for the secretion of hydrochloric acid, Troller[14] and Schreuer and Alfred Riegel have shown that no influence as to stimulation of secretion can be assigned to the saliva. Of course, the important physical action of the saliva (for example, the lubrication of the bolus) must not be disregarded.

As to what concerns the act of digestion proper, we have again to mention as most important the epoch-making investigations of Pawlow and his disciples. These have incontestibly proved to us that the secretion of the gastric juice in dogs depends regularly on the kind of ingesta. How far these results apply to human pathology cannot be estimated at the present time. According to the recent investigations of Schüle,[15] Troller, and Schreuer and Alfred Riegel,[16] they cannot be accepted without modification. For instance, according to these authors, as was, however, already known, the human stomach reacts with a secretion of hydrochloric acid upon the ingestion of egg-albumen, while, according to Pawlow, this is not the case with the stomachs of dogs.

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[13] Schreuer and Alfred Riegel, Zeitschrift für dietetische und physikalische Therapie, 1901, Bd. 4.

[14] Troller, Zeitschrift für klinische Medicin, 1899, Bd. 38.

[15] Schüle, Deutsches Archiv für klinische Medicin, 1901, Bd. 70, P. 112.

[16] Schreuer and Alfred Riegel, *l.c.*

Another method, leading directly to the goal, is that by means of systematic tests of the act of digestion in the healthy and sick, such as those with which v. Leube[17] and more recently Penzoldt[18] have to be credited.

Several years ago v. Leube sought to establish a kind of scale of the most easily digested up to the most difficultly digested articles of food. This diet, at first designated for the treatment of ulcer, is also available for other chronic gastric affections in which it is necessary to increase gradually the functional activity of the stomach. The diet is less adapted for a number of other conditions (for example, insufficiency of the stomach of the first and second degrees, chronic inacid or subacid gastritis). We give the celebrated four diet-lists of v. Leube as follows:—

1. Bouillon, meat-solution, milk, soft-boiled and raw eggs, sweetened zwieback free of fat, or cakes [crackers]. Water or natural acidulous waters, not rich in carbonic acid.

2. Boiled calf's brain, thymus gland [sweetbread], chicken, squab, rice or barley slime-soup, tapioca boiled in milk, finally boiled calf's feet.

3. Scraped sirloin steak, scraped raw ham, purée of potatoes, a little light bread, tentatively small doses of coffee or tea with milk.

4. Broiled chicken, squab, venison, partridge (rabbit is less to be recommended), roast beef (medium), roast veal (leg), pike, boiled perch, macaroni, rice-bouillon, later, light puddings, wine in small portions.

A second and rather useful idea rests in testing in healthy persons the digestibility of the greatest possible number of articles of food in a certain amount, form, and preparation, in order to determine a basis for the feeding of patients suffering from gastric diseases. This method has been advanced in a systematic manner for a number of years by Penzoldt and his adherents. Determination of the time at which the stomach had fully emptied itself after the ingestion of a certain amount of food or drink was always regarded as decisive, but here the chemical behaviour of the ingesta was also tested, and the motor function of the stomach served as a scale. The following table, quoted from the work of Penzoldt, serves to elucidate these points. There leave the stomach in

One to two hours.

100 to 200 gm. plain water.
220 gm. carbonated water.
200 gm. tea without addition.
200 gm. coffee without addition.
200 gm. cacao without addition.
200 gm. beer.
200 gm. light wines.
100 to 200 gm. milk, boiled (A 3 to 5, F 3 to 5, C 5).*
200 gm. meat-broth, without addition.
100 gm. eggs, soft.

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* A, Signifies albumen. F, Fat. C, Carbohydrate.

[17] v. Leube, Zeitschrift für klinische Medicin, 1883, Bd. 6.

[18] Penzoldt, Deutsches Archiv für klinische Medicin, 1893 and 1894, Bd. 51 and 53.

Two to three hours.

200 gm. coffee with cream.
200 gm. cacao with milk.
200 gm. Malaga wine.
200 gm. Tokay wine.
300 to 500 gm. water.
300 to 500 gm. beer.
300 to 500 gm. milk, boiled.
100 gm. eggs, raw and scrambled, hard-boiled, or as omelet (A 12, F 12).
100 gm. beef-sausage, raw.
250 gm. calf's brain, boiled.
250 gm. calf's sweatbread, boiled.
72 gm. oysters, raw.
200 gm. carp, boiled.
200 gm. pike, boiled (A 18, F 0 to 5).
200 gm. codfish, fresh, boiled (A 17, F 0 to 5).
200 gm. codfish, salted, boiled (A 80, F 1).
150 gm. cauliflower, boiled (A 2, C 4).
150 gm. cauliflower as salad.
150 gm. asparagus, boiled (A 2, C 2).
150 gm. potatoes, boiled in salt water (A 2, C 2).
150 gm. mashed potatoes.
150 gm. stewed cherries.
150 gm. raw cherries.
70 gm. light bread, fresh or old, dry or with tea (A 7, C 52).
70 gm. zwieback, fresh or old, dry or with tea.
70 gm. pretzels.
50 gm. Albert biscuits.

Three to four hours.

230 gm. spring chicken, boiled (A 20, F 4).
230 gm. partridge, boiled.
220 to 260 gm. squab, boiled.
195 gm. squab, broiled.
250 gm. beef, raw, boiled, lean (A 21, F 1 to 5).
250 gm. calf's feet, boiled.
160 gm. ham, boiled (A 24, F 36).
160 gm. ham, raw.
100 gm. roast veal, warm or cold, lean (A 20, F 1 to 5).
100 gm. roast beef, cold or warm.
100 gm. beefsteak, raw, scraped.
100 gm. tenderloin.
200 gm. salmon, boiled (A 16, F 28).
72 gm. caviar, salted (A 31, F 16).
200 gm. sardines in vinegar, kippered herring.
150 gm. brown bread (A 6, F 0 to 5, C 50).
150 gm. bran bread.
150 gm. light bread.
100 to 150 gm. Albert biscuits.
150 gm. potatoes.
150 gm. rice, boiled (A 31, C 76).
150 gm. kohlrabi, boiled (A 3, C 8).
150 gm. carrots, boiled (A 1, C 9).
150 gm. spinach, boiled.
150 gm. cucumber salad.
150 gm. radishes, raw.
150 gm. apples.

Four to five hours.

210 gm. squab, broiled.
250 gm. fillet of beef, broiled.
250 gm. beefsteak, broiled.
250 gm. beef's tongue, smoked (A 24, F 31).
100 gm. smoked beef, sliced (A 27, F 15).
250 gm. hare, broiled.
240 gm. partridge, broiled.
250 gm. goose, roasted (A 16).
280 gm. duck, roasted.
200 gm. salted herring.
150 gm. lentils, purée (A 25, C 54).
200 gm. peas, purée (A 23, C 52).
150 gm. string beans, boiled (A 3, C 6).

On this foundation Penzoldt, with the employment of the mentioned diet-lists of v. Leube, has formed a new dietary, which, through stating the amount of weight, the preparation and other qualities of the articles of food, advantageously distinguishes itself from v. Leube's list, but which unluckily is likewise suggested only for a diet in ulcer. So, for example, vegetables, puddings, and fats, which are preferably to be considered in the diet in the treatment of gastritis and superacidity, and partly also of carcinoma, are rather neglected in the diet-lists of Penzoldt. Nevertheless it doubtless offers valuable hints for the treatment of ulcer as well as for the so-called "nursing treatment of the stomach" in general.

Penzoldt's dietary is likewise made up of four lists, as shown on pages 283 and 284.

Many difficulties lie in the way of our employing these fundamental lines of a diet-list in a given case. We must consider the life-habits of the patient, his inclinations and disinclinations for or against certain articles of food, his wishes for change in cuisine, the successive alterations according to change of condition of health, etc.

We may take it as a principle of practice to order for our patients from suitable articles of diet the so-called most digestible ones. To be sure, the term "digestibility" is very indefinite, yes, in general it may be said, as I pointed out many years ago in an article,[19] that food substances cannot be designated at all as easy or difficult to digest. On the other hand, the implied meaning of "digestibility" of certain articles of food can hardly be misconstrued. We know that cabbage, fats difficult to melt, spicy and acid substances, and alcohol in large quantities trouble the digestive functions to a greater or lesser degree. Only in certain cases, namely, in atony of the intestinal canal *and intact function of the stomach* we may depart from this experience without danger, because in just such cases the undigested and indigestible food-remnants, as well as the substances forming fermentation, may furnish a favourable stimulus to the diminished reflex activity of the intestinal canal.

The better we are informed concerning the subjective and objective disturbances of the work of digestion in special, the better we shall be able to choose the most suitable food for the patient in making up the *special dietary*. Here it must especially be noted that the diet must take into consideration not only the disturbed gastric functions, but also—it does not matter whether they be affected simultaneously or not—the functions of the intestines; thus, for example, gastric disturbances are often associated with habitual constipation, or, on the other hand, with habitual diarrhœa. In the first a simple "nursing" diet, that would be free from chemical or mechanical stimuli of the intestinal peristalsis; would help the stomach, but would absolutely inhibit intestinal action. Habitual diarrhœa, on the other hand, requires a special diet for the intestine, apart from the "nurs-

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[19] Boas, Berliner klinische Wochenschrift, 1890, No. 20-23.

Diet I (about 10 days).

FOOD OR DRINK.	LARGEST AMOUNT AT ONE TIME.	PREPARATION.	SPECIAL REQUIREMENTS.	HOW TO BE EATEN.
Meat broth.	250 Gm. (1/4 litre).	From beef.	Without fat, not salted, or only a little.	Slowly.
Cow's milk.	250 Gm. (1/4 litre).	Well boiled, or sterilized (Soxhlet's apparatus).	Entire milk (or lime-water 1/3, milk 2/3).	If desired, with a little tea.
Eggs.	One or two.	Very soft, just heated or raw.	Fresh.	If taken raw should be stirred into the warm, not boiling meat-broth.
Meat solution. (Leube-Rosenthal).	30-40 Gm.		Should have only a slight meat-broth odor.	In teaspoonful doses or stirred into the meat-broth.
Crackers (Albert biscuits).	Six.		Without sugar.	Not softened, but well chewed and insalivated.
Water.	1/8 litre.		Ordinary water, or natural carbonated water with a small percentage of CO_2 (Selters).	Not too cold.

Diet II (about 10 days).

FOOD OR DRINK.	LARGEST AMOUNT AT ONE TIME.	PREPARATION.	SPECIAL REQUIREMENTS.	HOW TO BE EATEN.
Calves' brain.	100 Gm.	Boiled.	Freed from all membranes.	Best taken in meat-broth.
Calves' thymus.	100 Gm.	Boiled.	Likewise, especially carefully isolated.	Best taken in meat-broth.
Pigeon.	One.	Boiled.	Only young, without skin, tendons, and the like.	Best taken in meat-broth.
Chicken.	As large as a pigeon.	Boiled.	Only young, without skin, tendons, and the like (small fattened chicken).	Best taken in meat-broth.
Raw beef.	100 Gm.	Chopped fine, or scraped, with only a little salt.	From the tenderloin.	To be eaten with crackers.
Raw beef-sausage.	100 Gm.	Without addition.	A little smoked.	To be eaten with crackers.
Tapioca.	30 Gm.	With milk, cooked to make gruel.		

Diet III (about 8 days).

FOOD OR DRINK.	LARGEST AMOUNT AT ONE TIME.	PREPARATION.	SPECIAL REQUIREMENTS.	HOW TO BE EATEN.
Pigeon.	One.	Broiled with fresh butter, not too much seasoned.	Only young, without skin, tendons, and the like.	Without gravy.
Chicken.	One.	Broiled with fresh butter, not too much seasoned.	Only young, without skin, tendons, and the like.	Without gravy.
Beefsteak.	100 Gm.	With fresh butter, rare. (English style.)	The meat from the tenderloin, well beaten.	Without gravy.
Ham.	100 Gm.	Raw, scraped fine.	Weakly smoked, without the bone.	With white bread.
Milk-bread, or Zwieback or Freiburger [Lititz] pretzels.	50 Gm.	Baked crisp.	Stale (so-called rolls, etc).	Carefully chewed, well insalivated.
Potatoes.	50 Gm.	(*a*) Mashed. (*b*) boiled in salt water.	The potatoes should be mealy, and should crumble on crushing.	
Cauliflower.	50 Gm.	As a vegetable, boiled in salt-water.	Only the flowers are to be used.	

Diet IV (about 8 to 14 days).

FOOD OR DRINK.	LARGEST AMOUNT AT ONE TIME.	PREPARATION.	SPECIAL REQUIREMENTS.	HOW TO BE EATEN.
Venison.	100 Gm.	Roasted.	Saddle, not too fresh, but without "haut-gôut."	
Partridge [quail]	One.	Broiled without bacon.	Young animals without skin, tendons, legs, etc; should hang for a time.	
Roast beef.	100 Gm.	Medium to rare.	From good, fatted cattle; well-beaten.	Warm or cold.
Tenderloin.	100 Gm.	Medium to rare.	From good, fatted cattle; well-beaten.	Warm or cold.
Veal.	100 Gm.	Roasted.	From good, fatted cattle; well-beaten.	Warm or cold.
Pike, shad, carp, trout.	100 Gm.	Boiled in salt-water, without addition.	All fish-bones should be carefully removed.	In the fish-gravy.
Caviar.	50 Gm.	Raw.	Salted a little. (Russian caviar.)	
Rice.	50 Gm.	Mashed, pushed through a sieve.	Soft boiled rice.	
Asparagus.	50 Gm.	Boiled.	Soft, without the hard part.	With a little melted butter.
Scrambled eggs.	Two.	With a little fresh butter and salt.		
Omelet (soufflé).	Two.	With 20 Gm. sugar.	Must have risen well.	To be eaten at once.
Stewed fruit.	50 Gm.	From fresh boiled fruit, to be strained through a sieve.	Free from all kernels and peel.	
Red wine.	100 Gm.	Light, pure, Bordeaux.	Or some corresponding kind of red wine.	Slightly warm.

ing" diet. In such cases, a milk diet, for instance, or a diet rich in sugar would be a gross dietetic error.

As is to be seen, directions for diet, to be successful, must be associated with a thorough instruction of the patient. This must consist in

1. An exact statement of time for the separate meals.
2. An exhaustive list of the allowed nutrients and refreshments.
3. A fairly exact direction as to the weight and the amount of the foods and drinks.
4. Short directions as to the preparation of the foods, temperature of the drinks, and any other pertinent remarks.

As to 1: A regulation of the time for each meal is to be desired, because the cause, or at least the occasion, of the digestive disturbances may lie in a false division of time for ingestion. In general, it may be held as a rule to advise small and frequent meals, but this is not useful or at all necessary in all cases. For the frequent ingestion is associated with a disadvantage not to be disregarded, namely, the absence of pauses for the rest of the diseased organ. The latter are to be considered especially in cases of abnormally increased glandular secretion. In general, the frequent and small meals are adapted in all cases of atony of the stomach and stenosis of the pylorus, in which the musculature of the stomach manages small quantities well, and becomes insufficient only with the larger meals. But also in the first-mentioned cases an exact time division is desirable in order not to induce overloading on the one hand, and on the other hand frustrate secretion of the gastric juice, with its unfavourable effects upon the mucous membrane of the stomach.

As to 2: As a rule this point is commonly disregarded, according to my experience. More is usually forbidden and less allowed than is good for the patient. I am of the the opinion that it is the duty of every physician to question as to whether any dietetic restriction applies to the exigencies of the case. The objection that we may injure more by great compliance than by the contrary does not hold good, as we may also injure by too great a deprivation of the whole organism, and in many cases take from it the possibility of finding a compensation for the already weakened and diminished assimilation. Also it must not be overlooked that by too great a limitation of the allowed foods a certain monotony, indeed an aversion to the cuisine, may be established, which asserts itself either in temporary anorexia, or, still worse, in an explosive violation of the dietary restrictions, and thus increases the trouble.

In dietary directions, shall we give rules as to the forbidden foods and drinks? Here I deviate from the prevailing customs, and that on the following grounds: General prohibitions (for example, the well-known advice, to eat nothing acid, fat, or spicy), are worthless as a whole, because the patient does not know what to do with them, and above all, because this generalisation goes actually too far. Detailed directions about *all* forbidden substances would be of importance if they only were actually complete. But even the strictest carefulness of the physician cannot make them so. The patient may, if he will, make the most pronounced dietetic errors even in spite of the most specific prohibitive regulations. This leads to the partly comic, partly aggravating controversies between physician and patient, which are best avoided by designating *only what is allowed* and adding the remark, best in writing, *"what is not allowed is forbidden."*

As to 3: The importance of exact quantities of the ingesta needs hardly any comment. This is not difficult when one has clear ideas as to the amount of food and drink necessary in the healthy. In this respect, there is no need for us to be too pedantic and to carry the conscientiousness to scruples; but we should not choose such units as a cup, wine-glass, tablespoonful, plate, which naturally vary within the widest limits.

As to 4: Directions as to the methods of preparation of the ingesta are commonly necessary. Sometimes the patient himself asks for explicit directions. The remarks should deal with the preparation of meat (raw, under-done, well-done), as to whether gravies should be served with them (an important point), and the way to prepare the latter; further, with the artificial division of the meat (chopping, mincing, etc); finally, with the addition of condiments, (salt, pepper, paprika, mustard, lemon, nutmeg, vinegar, oil, etc.). The same answers also for fish. As to vegetables, farinaceous food, preserves, etc., the question of preparation is also of importance; in the former particularly, the mechanical division, then the choice of additions (fat, sugar, condiments, acids, etc.); as to preserves, the way of serving in the original form, as marmalade, with the addition of sugar or acid, or other similar substances.

As to drinks and liquids (soup, coffee, tea), we must consider first of all the temperature.[20] We give here a table of the most desirable temperatures of the most important drinks and foods:[21]

	Temperature of article.
Water	+ 12 to 13° C.
Selters and soda water	+ 10 to 12° C.
White wine	+ 10° C.
Red wine	+ 17 to 18° C.
Beer	+ 12 to 15° C.
Coffee and tea, not over	+ 40 to 43° C. (Wiel)
Meat broths (milk and flour soups)	+ 37 to 43° C.

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[20] Compare the instructive treatise by Uffelmann, Ueber die Temperatur unserer Speisen und Getränke, Wiener Klinik, 1887, Heft 6.

[21] According to Munk and Ewald. Ernährung des gesunden and kranken Menschen, 1895, third edition, P. 320.

	Temperature of article.
Milk (not under 16 to 18° C.), not over	+ 33 to 40° C.
Purées (porridges)	+ 37 to 42° C.
Roasted foods	+ 40° C.
Bread, not over	+ 30° C.

As to spiritous liquors, the amount of alcohol and sugar is of importance, and the amount of carbonic acid for mineral waters. For tea, coffee, and cocoa, directions as to the strength of the infusion, and whether substances (sugar, milk, rum, cognac) should be added is of importance.

Our dietary therapeutic task is, however, not yet exhausted. However well planned the diet may be, it will only exceptionally fulfil all the indications of the disease. It happens that patients, contrary to the rule, cannot tolerate this or that food, and sometimes have an aversion to a food.

Above all, however, the course of the disease, from week to week, requires changes, and thus the diet may need additions or subtractions, and, according to the condition of the weight, an increase of the constituents rich in calories or (when there is an abnormal increase in weight), a reduction; or the diet may need modifications in various directions, according to whether the effect on the intestine is favourable, excessive, or unsatisfactory.

As we see, a dietary direction given *"lege artis"* is a complicated and, particularly for the young physician, a difficult undertaking, especially as the medical student had not been instructed in this subject (in contrast to writing prescriptions). Our modern age of machinery has invented a substitute in the institution of a dietetic "calender" (the leaves of which can be torn off). It is a pity that such a mischief should be practised and also defended by scientifically versed physicians, the same physicians who cannot accentuate enough a strictly individualised treatment. How is such a "pattern" diet congrous with the spirit of an individualising treatment? Hence, "away with printed or otherwise manifolded diet slips." Each patient must receive his own diet-list, adapted to the characteristics of his case, the condition of his nutrition, his habits of living, etc. Let him for whom this is too much trouble seek other fields than that of medicine.

Entirely different is the question as to whether it is advisable that the physician should be instructed in the "prescribing" of a diet list. This question must be answered in the affirmative. Prominent clinicians, like v. Leube, Penzoldt, etc., have a perfect right to give such diet-lists as may serve for the use and the guidance of the physician; but *they serve only as paradigmas, not for thoughtless copying.*

As far as diet in the individual affections is concerned it is not easily possible nor requisite to give more than a general review at this place.* Let us begin with the most harmless disturbance, *acute gastritis.* The indications here are quite evident; they read simply: sparing of the affected organ. Here we will be able to renounce the administration of a diet rich in calories, and that the more so because the loss of weight due to underfeeding is soon compensated by a quickly improving appetite and the increased ingestion of food. The principle that only fluid, non-irritating (slimy) food is most suitable has been substantiated by thousandfold experience. For the beginning of a diet richer in calories we must be governed by the condition of the tongue, a genuine longing for food, and finally the cessation of the subjective symptoms and of a fever, if the latter was present.

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* The special directions are to be found in the Special Part of this work, in the chapters on the various diseases.

Just as clear are the indications in severe disturbances of motility due to stenosis. Before all, in this case, the substances must be given in such a form as will cause no difficulty in their passage through the narrowed pylorus. Here, the most ideal food would be the liquid. This has been substantiated by practical experience in severe cases of pyloric stenosis. One extraordinarily important objection, however, is the fact that it will not be sufficient for the improvement or maintenance of the organism. We must, therefore, always endeavour to give not only liquid foods, but also small quantities of pulpy or semi-solid substances. Here, however, the secretion of gastric juice plays a deciding rôle. If this is sufficient, as it is in benign pyloric stenosis, and sometimes also in malignant, we can give albuminous substances well prepared, in solid though finely divided form, namely, the lighter kinds of meats and fish, of carbohydrates those that can be served as purées, and of solid fats, small quantities of butter (about 50 to 60 grammes a day). On the other hand, the diet in pyloric stenosis with no secretion at all, or almost none, is quite different—the albuminous bodies insoluble in water are either not digested at all, or are digested only to a slight extent, and it is our duty to administer the albuminous substances necessary for maintenance in a liquid vehicle. The same is also true of carbohydrates. The former is brought about by admixtures of albumen and yolk of egg in soups, albumoses, peptones, meat-powder, meat and fish purées, and the latter as concentrated amylaceous or leguminous soups, soups containing dextrine, of which we have a great choice. (See page 298.) Here also a slight amount of butter may be used, likewise in soup. Patients with pyloric stenoses must eat frequently and a little at a time. On the other hand, periods of rest, such as are gained by an early and the smallest possible evening meal, are very desirable. In such cases washing-out of the stomach in the evening may be of benefit.

Important also is the postulate, brought forward on many sides, to let such patients, as stomach patients in general, keep quiet during digestion. That the patient should lie on the right side in order to facilitate the exit of chyme has been correctly emphasised. According to my experience, however, this position is not always well borne.

A considerable influence is exerted upon the diet in such cases by the fermentative and putrefactive processes always associated with pyloric stenosis. The more, however, we succeed by a rational diet in overcoming the disturbance in passage—in other words, the more we bring about thorough evacuation of the stomach within the normal time after ingestion of an accurately regulated diet—the less frequently will fermentations in the stomach occur. To the latter, which formerly always stood in the foreground of consideration, less attention has to be paid than to the emptying of the stomach within the right time. How important the systematic measuring of the gastric contents obtained from the fasting stomach, as has been carried out at my clinic for years, is for the judgment of this state of affairs, needs no special mention.

The question of feeding in atony is similar to that in stenosis—we know to-day that liquid substances undoubtedly leave the stomach quicker than solid substances; it should here also be our aim to administer the substances in a purée or in a fluid form; and the more especially in fluid form, the more deficient the secretion of the stomach. Here also the dictum holds good—never any overloading of the stomach!

Conçerning the further simple disturbances of secretion not complicated with mechanical insufficiency, views are still divided as to the dietetic measures. In superacidity, for example, theory requires greatest possible restriction of the amylaceous foods and pronounced recommendation of much meat and proteids in general, on the assumption that hereby the excess of hydrochloric acid is thus bound up and its irritating action on the gastric mucous membrane avoided. On the other hand, there are also many opinions which, on the other side, ascribe an irritation on the gastric secretion to the free ingestion of meat and albumen, and recommend an amylaceous diet. This standpoint, which for many years has had an adherent in v. Sohlern,[22] has been made the centre of the discussion, by the recent labours of Jürgensen,[23] in Copenhagen. Jürgensen pleads, by reason of his experience as well as of the experiments of v. Sohlern and v. Jaksch on healthy persons, and which were confirmed by A. Schüle,[24] for a predominant vegetable diet in increased secretion of gastric juice. Jürgensen's communications offered the opportunity of examining the question in all directions, and the existing material may be considered as sufficient for a critical judgment.*

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* Compare Part II, page 693, *et seq.*

[22] v. Sohlern, Berliner klinische Wochenschrift, 1891, No. 20-21.

[23] Jürgensen, Archiv für Verdauungskrankheiten, 1897, Bd. 3, P. 2.

[24] Schüle, Zeitschrift für klinische Medicin, 1895 and 1896, Bd. 28 and 29.

Accordingly, we may take the stand that a predominant meat-diet for patients with superacidity must be regarded as not well adapted, and that, further, carbohydrates in a suitable form are to be recommended for the feeding of such patients. We cannot, however, go so far, as to prescribe a strictly vegetable diet to patients with superacidity, first, because the same encounters great doubt as to the necessary nutritive value; then because the acidity of the gastric juice is not permanently diminished in spite of continued administration of carbohydrates; and finally, as experience teaches, because the ingestion of meat in moderate amount is well borne in superacidity. A mixed diet, therefore, is the most suitable one also in such cases.

Far more important than the question of carbohydrates, is the question of fats. In this respect the investigations of Ewald and Boas, Akimow-Peretz, Strauss and v. Aldor, Backmann and Buch have brought forward evidence that fats inhibit the acid secretion, at least temporarily, and even perhaps permanently, as Buch and Akimow-Peretz assert, and that they are exceedingly well borne and assimilated. In any case proof is furnished that fats in appropriate form are eminently adapted for the treatment of superacidity.

It must be understood that these remarks apply only to uncomplicated superacidity, whether the same is the precursor of an ulcer or a symptom of gastritis acida, or, finally, of a purely nervous origin. When, however, a motor insufficiency is co-existing, the standpoint of emptying of the stomach is so prevailing that the consideration of acid secretion, which essentially is dependent on the insufficiency, is subordinated.

Still other indications are existing in superacidity associated with *ulcus ventriculi.* Here the most important aim is to exclude from the diet all mechanical, chemic, and thermic irritations and harmful influences, whereby the preference of vegetable to animal substances again is relegated to the background. Of course, the former, in form of soups and purées, have the preference, because they leave the stomach as quickly as possible, while meat, even when prepared very suitably, always induces a certain secretion of gastric juice.

The opposite functional anomaly, the disappearance of the secretion of gastric juice, *achylia gastrica,* as it was first called by Einhorn, with its various intermediate stages requires the following dietetic consideration. The albuminous bodies, especially meat, are to be taken in normal amount, but must be of selected quality and suitable preparation; artificial albuminous preparations are hardly necessary. Martius,[25] to be sure, believes that we can also give a heavier meat diet, and he has satisfied himself as to their excellent digestion. There is no doubt that this is possible with good duodenal digestion, but it is questionable as to whether it is advisable to burden the intestine with the work, which normally it does in conjunction with the stomach. We believe, therefore, in the integrity of the above-mentioned principles. Very well digested are the vegetable substances in such cases, as far as they may be ingested in the form of purées, and it must be our task to introduce them into the diet in the form of amylaceous soups, vegetable purées, amylaceous food and puddings, and also preserves.

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[25] Martius, Ueber Achylia gastrica, Wien and Leipzig, 1897.

It is important to mention that the fats in a good form, in the first place butter-fat, are, apart from individual exceptions, excellently borne in these types of secretory disturbances. The presumption of these dietary directions is that the motility of the stomach is good and the pylorus not obstructed. When achylia gastrica is complicated with disturbed motor function and pyloric obstruction, the basis of diet which we have already pointed out holds good.

It is rather difficult to determine the diet in *carcinoma.* The decisive factor in this case is the presence or absence of a stenosis of the stomach, that is, whether retention is or is not present. In the latter case the diet may keep entirely within the limits just mentioned for the diet in chronic gastritis. In such cases, the appetite is, above all, the determining factor. When the latter fails, we must, in order to keep the patient above water, drop all scholastic views concerning easy and difficult digestibility and must experiment with this or that food. How easy it is to make such patients happy through some harmless relish, and only absolutely harmful articles of food (such as mayonnaise, bacon, salads, cabbage, etc.) are to be banished energetically. The view of Beneke and others, brought forward in the seventies of the last century, concerning the favourable influence on carcinoma exerted by a vegetarian diet has not been proved in practice, yet in the majority of cases the same is preferred to animal diet. That, however, the carcinoma in itself has nothing to do with the matter, is to be seen, for instance, in patients who, after a successful gastroenterostomy, return again to a mixed diet as they did when healthy, and progress remarkably.

Concerning *disturbances of absorption* as symptoms of stomach diseases, our knowledge to date is indeed only too meagre. It seems, according to our experiences, that the occurrence of genuine independent disturbances in the lymph-apparatus of the stomach is very doubtful. For D. Gerhardt[26] as well as especially v. Mering,[27] and later Strauss,[28] found that even in the most severe stomach diseases (apepsia gastrica, carcinoma) sufficient material may still be absorbed. Although this primarily holds good only for carbohydrates, we may assume that the absorption of peptones can hardly be otherwise.

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[26] D. Gerhardt, Berliner klinische Wochenschrift, 1898, No. 35.

[27] v. Mering, Klinisches Jahrbuch, 1899, Bd. 17.

[28] Strauss, Zeitschrift für klinische Medicin, 1900, Bd. 41.

It follows from this that in our dietary regulations we need not take especial account of absorption disturbances.

Sensory disturbances in the sphere of the stomach are either the expression of an organic disease, or a symptom of a so-called nervous dyspepsia in its various manifestations.

In many cases, especially of the first group, we are able to steer clear of discomfort manifested during digestion by means of suitable directions; thus by strict regulation of the temperature we may prevent the occurrence of gastralgias that follow the ingestion of cold drinks. In other cases, where similar manifestations are due to the ingestion of highly-spiced or acid foods, we can likewise find the remedy in prohibiting the same.

It is different in *nervous dyspepsia* and its related forms. Every calculation fails in these cases. It happens that substances at one time well borne, yes, even excellently borne, may cause at other times the most disagreeable symptoms. To these belong by no means, as we might believe, only foods usually considered difficult of digestion, but, quite the reverse, such substances as have been generally proved to be easily digested. In these cases a cautious experimentation is indispensable.

As far as my observations have taught me, in all these cases of a capricious and moody stomach a varying and at the same time copious diet is an essential condition for well-being; milk and abstinence cures are never less suitable than in neurasthenia of the stomach. Restriction of fats ought to be particularly avoided.

The same holds true for the protean forms of "nervous vomiting." We can best find out what the stomach can bear, by cautious and systematic experimenting. As a principle for all cases of nervous vomiting the following holds: avoidance of large quantities of liquids, especially during and immediately after each meal.

Disturbances of a single function are rare in chronic diseases of the digestive apparatus; we discussed above the anomalies of the individual functions only for the sake of a schematic regulation. The consideration of all clinical factors is indispensable for the feeding in individual cases. Likewise the statements made are true only for cases of idiopathic disturbances of the stomach; while in cases of secondary impairment of the functions of the digestive apparatus, the dietetic consideration of the *primary affection* is necessary. Without doubt here also the knowledge of the condition of the digestive functions is not to be underestimated.

To the results of objective examinations belong also *investigations of metabolism*, in as far as they teach us whether the individual components of the foods become assimilated and manifest themselves in the body as meat or fat addition, or whether, for any reason whatever, they remain unused and leave the body, or whether they are destroyed in the body in spite of good absorption. Systematically carried out investigations of metabolism of this kind would be of great value, and they would give answer in this or that direction as to the slightest variations in metabolism, more precisely than the scales can do. Unfortunately they are still too complicated to find employment at the sick bed.

A further method that occasionally yields information and may serve as an index for our dietetic treatment, is the examination of the fæces. Especially easy is it to find in the stool remnants of connective tissue and undigested muscle-fibres in severe catarrhs of the stomach, as soon they are associated with catarrhs of the intestine (Biedert, M. Einhorn, B. Oppler, A. Schmidt, Boas, H. Strauss, Zweig, and others), and sometimes entirely undigested pieces of meat of considerable size are seen. As a matter of course, a small quantity of muscle-fibres occur normally, particularly after ingestion of a copious quantity of meat. On careful examination of normal and abnormal fæces (best by means of the now well-recommended stool-sieves), and with consideration of the last-mentioned factor, a judgment is possible as to the pathological increase of the muscle-fibres, if repeated investigations are equally positive.

In similar manner, tests of the fæces yield us information as to the possible incomplete digestion of starch (tests with iodine-potassium-iodide, sugar-tests) or digestion of fat (estimation of fat, fatty acids, and soaps, according to the usual rules), and thus may give us useful points as to the necessity of a possible restriction of these foods.

Artificial Food Preparations.

The fact that proteids remain frequently undigested or are only partly digested in the stomach has already given an opportunity for the chemists and physiologists to produce preparations which are artificially prepared in such a way that they may be directly absorbed. Similar attempts have been made to prepare carbohydrates in a particular manner so as to convert the same into a readily soluble form (usually powder form), free from waste. More recently also, they made the attempt of bringing fats into a form more adapted for digestion.

As to the artificially predigested *proteids*, they are prepared by various methods, partly through artificial digestion by means of the well-known enzymes (pepsine, papain, pancreatin, [bromalin-pineapple]), partly through the influence of over-heated steam. In some preparations the manufacturing process is not known. Artificial products of this kind are called peptones, or, more correctly albumoses.

In the course of the last two decades a great number of such products have been put on the market, and particularly within the last ten years great progress has been made in improving the taste and the concentration of these albumoses. Furthermore, it may be considered as proved that the albumoses are assimilated in the body similarly to meat, and that therefore the same may be regarded as a substitute for egg-albumen, meat or fish.

The practice seems to indicate that such products are in great demand, and that the general public, seduced by the extraordinary promises in the daily newspapers, urgently demands their use, in the hope of gaining new life and new strength from them. Exact science must not be misled; it must, uninfluenced by the daily advertisements, figure on the real value of these food preparations, and, if the results of objective investigations demand it, warn against their over-estimation.

The first question is: is there a necessity for such artificial products, and, if so, for which conditions? Such a necessity may arise under three circumstances. First, when a natural peptonisation, or rather albumosisation, does not occur. This happens most frequently in cases of chronic glandular gastritis, then in cases of carcinoma of the stomach, and also permanently or only temporarily in other gastric affections.

In chronic gastritis, in which the necessity of such substitutes ought to be usually felt the most, there is, as experience shows, never any reason for it. If the patients have appetite, they tolerate also natural albumen and its products, provided they are prepared in the right manner. If there is loss of appetite, artificial products will not be capable of replacing the proteid deficit in the body to any sufficient extent. The case is similar in cancer of the stomach. We cannot help much with the few grammes of albumen which we administer the patient in the form of albumoses; the patient himself recognises this fact only too soon, and the food preparations, upon which he has put all his hopes to-day, he will abandon to-morrow. Moreover, the administration of large doses of albumoses is usually impossible—there are, of course, exceptions—on account of the high price, the bad taste, and also on account of rapidly appearing dislike and other disturbances (diarrhœa).

Secondly, albumoses may be indicated in cases where we intentionally wish to relieve the stomach and to introduce food which, being soluble in water, is particularly adapted for ready admission into the intestines. Such an indication exists, for instance, in gastric ulcer, and also in motor disturbances of a high degree, particularly in pyloric stenosis, whatever its origin may be. Indeed, the administration of Leube-Rosenthal's meat-solution is still approved by many physicians as an aid in the nutrition in gastric ulcer. Less approved is the administration of albumoses in pyloric stenosis, although in just these cases where the hydrochloric acid secretion is lost, an assistance to the nutrition is not without value.

Finally, we might think of administering albumoses as an aid to the nutrition in general, namely, in cases where a debilitation of the body has been caused by the disease itself or its consequent diet. This indication is the least well-founded, but, as Klemperer[29] has stated so well, the nutritive value of the highly concentrated albumoses is nevertheless so slight that it cannot compete at all with our usual *natural* food-substances, particularly the egg-albumen or the milk.

A little better than with the artificial mixture of albumoses is the effect with the so-called *meal-powders* and *leguminoses*, an addition of which to soups is capable of increasing the nutritive value of the same, though it may not be considerable, particularly if we take care to prepare thick and concentrated soups [gruel]. We need not take into special account the amount of dextrines in some special products, such as Nestlé's Food, Kufeke's Food, [Horlick's Malted Milk, Malted Food, Eskay's Food], as a natural saccharification of the starch is rarely entirely lacking even in grave gastric disorders.

Finally, the artificial *fat-preparations* are intended for introducing fat in a most appropriate form into the stomach. Some writers took particular pains to manufacture oil rich in fatty acids, in order to relieve the intestinal juices from the task of fat-splitting, or to facilitate the absorption of fat where the same is defective for any reason. The oldest of fats of this kind is the cod-liver oil; the more recent, the lipanin and v. Mering's Kraft Schocolate [Hauswaldt Vigor Chocolate], and Lipogen-chocolate. The ingestion of large amounts of fat is particularly recommended for the improvement of the state of nutrition, but, besides this, subjective tolerance comes into account.

Without doubt, a great increase of adipose tissue can be obtained by means of the mentioned fats as well as of the natural fats, whereby the organism may be supported; but the question is, whether we cannot obtain the same results by means of the neutral fats which nature offers us, such as butter, cream, and oil. The latter must certainly be answered affirmatively; we shall therefore make use of artificial fats only when a special cause speaks against the employment of the natural fats.

The discussions on this subject teach us that, on an average, the value and importance of the artificial food-preparations is often overestimated. They deserve occasional employment, but, as a whole, their indication is limited. The desire for change, or the very demand that something be done, brings, under some circumstances, the employment of such artificial methods to our mind; but the physician ought to look out that the expectations of the patients, and particularly of their relatives, are not stretched too high. On account of the high prices, artificial food-preparations, especially of the albumoses and fats, are to be reserved only for well-to-do patients; while the same effect can be obtained for the poorer classes by means of appropriate preparation of natural foods, and that with considerably less expense.

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[29] Klemperer, Berliner klinische Wochenschrift, 1897, No. 26.

The acme of the era of artificial food-preparations seems rather to have passed; numberless preparations recommended, or rather thrown upon the medical and lay public by the clamor of advertisement, are already part of history, and only a few—and these not always the best ones—have maintained themselves in the favour of the general public and the physicians. How long will they last?

In conclusion, we give a brief review of the most-used preparations of albumoses, amylum, and fat.

*Preparations of Albumoses.**

Leube-Rosenthal Meat Solution: 1.8 to 6.5 per cent. peptones, besides 9 to 11 per cent. soluble albumen.

Kemmerich's Peptone: 30 per cent. albumoses, 18 per cent. peptones (Kühne), 9 to 12 per cent. soluble proteids; besides 8 to 10 per cent. potassium phosphate, potassium chloride, and earthy phosphates, 0.3 per cent. fat.

Koch's Peptone: 18.8 per cent. peptones, 16.0 per cent. propeptones, 1.4 per cent. insoluble albumen, 16 per cent. other nitrogenous substances, 0.8 per cent. fat, 6.9 per cent. salts, and 40.1 per cent. water.

Denayer's Peptone (mixture of albumoses and peptones): 19 per cent. organic substance, 10.5 per cent. albumoses, 1.5 per cent. peptones.

Somatose: tasteless and odourless powder, mixture of albumoses made of meat, soluble in water; contains 80 per cent. albumose.

Eucaseïn (sodium caseïn): white powder, readily soluble in water, tasteless and almost odourless; contains about 85 to 90 per cent. albumen.

Puro of Dr. Scholl, Meat Juice: 33 per cent. proteids, of which 21 per cent. is native albumen, 3 per cent. gelatine, 7 per cent. peptones (albumoses).

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* An excellent and complete compilation of artificial food-preparations is in the writing of Max Heim, Die künstlichen Nährpräparate und Anregungsmittel, Berlin, 1901.

Tropon, made by Finkler: 90 to 97 per cent. albumen, 0.5 to 1.0 per cent. ash, 0.1 to 0.8 per cent. ether extract. [A most excellent preparation, an odourless and tasteless powder, ready to be mixed with other food; may be used for making wafers, cakes, etc. Iron-tropon to be recommended.]

Soson: a finely powdered, light-yellow powder, insoluble in water, odourless, made of meat; contains 92.5 per cent. albumen, 3.3 per cent. water, 0.85 per cent. ash. [Likewise a good preparation.]

Roborat (vegetable albuminous preparation): 83 per cent. albumen, 11.9 per cent. water, 2.91 per cent. substances soluble in ether, 1.25 per cent. ash.

Plasmon (milk albumen preparation): 74.54 per cent. albumen, 12.56 per cent. water, 1.76 per cent. fat, 8.39 per cent. ash, 2.75 per cent. carbohydrates.

Sanatogen (casëin preparation); 95 per cent. casëin and 5 per cent. glycerophosphate of sodium.

[Mulford Predigested Beef: proteids (in form of peptones and albumoses) 4.58 per cent.; total solids, 10.82 per cent.; mineral matter (salts), 0.218 per cent.; alcohol (by weight), 17.175 per cent.

Valentine's Meat Juice: water, 54.40 per cent.; organic matter, 31.84 per cent.; ash, 13.75 per cent.; soluble albumen, 0.44 per cent.; alcohol extracts, 26.32 per cent.

Mosquera's Beef Meal (prepared by digestion with pine-apple juice): fat, 13 per cent.; proteids, 77 per cent.

Mosquera's Beef Jelly: 3 per cent. of insoluble material; of the soluble portion, 53 per cent. consists of albuminoids, which are nearly all peptones.]

2. *Carbohydrate Preparations.*

The most common infant's foods are to be found in the table on page 298, according to A. Stutzer.[30]

Furthermore, there are the following carbohydrate preparations in use:—[31]

Hartenstein's leguminous flours (four mixtures, according to the proportion of the nitrogenous and anitrogenous substances, and this pro-

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[30] A. Stutzer, Nahrungs- und Genussmittel, Weyl's Handbuch für Hygiene, Jena, 1894, Fischer.

[31] Nach Penzold-Stintzing, Handbuch der spez. Therapie, Bd. 3, S. 257.

	Nitrogenous Substances	Fat	Carbohydrates		Cellulose	Ash	Phosphoric Acid	Lime	Water
			Soluble	Insoluble					
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.
1. H. Nestlé, of Vevey	9.91	4.46	42.37	35.04	0.33	1.74	0.59	0.32	6.15
2. Faust & Schuster, of Göttingen.	10.79	4.55	43.21	32.99	—	1.92	0.51	—	6.54
3. Frerichs & Co., of Leipzig	11.96	6.02	28.76	44.48	—	2.36	0.52	—	6.42
4. Kufeke's Kindermehl . . .	12.51	1.81	21.92	52.22	0.65	2.11	0.63	0.11	8.78
5. Rademann, of Frankfurt .	13.62	5.37	15.51	55.51	0.82	4.06	1.72	1.04	4.54
6. Ridge, of London . . .	8.70	1.38	5.79	75.75	0.68	0.64	0.29	0.06	7.08
7. Carnrick, of NewYork. . .	16.69	5.53	28.11	41.32	0.18	3.00	0.87	0.64	5.17
8. Matthinson's Food, of Heasby . . .	0.20	—	70.50	—	—	0.90	—	—	28.40
9. Muffler & Co., of Freiburg.	15.10	5.10	32.37	39.78	0.10	2.43	1.32	1.00	4.76

portion in mixture I = 1 : 2.3, in mixture II = 1 : 3.3, in mixture III = 1 : 3.9, in mixture IV = 1 : 4.8).

Knorr's prepared flours (oats, barley, rice, lentil, groats, and prepared barley, etc.).

Liebe's leguminous flours (soluble only partly in water, contains much starch).

Mellin's food (is said to be entirely free of starch, fairly soluble, contains much sugar, but also particles which assume bluish colours on addition of iodine).

Timpe's preparations: (1) soluble leguminose, (2) soluble oatmeal, soluble only partly; besides Timpe's milk powder containing little starch and maizena (cornstarch).

Dr. Teinhardt's hygiama (reddish powder of slight cocoa taste).

Weibezahn's preparations (fine oatmeal and other preparations).

To the starch preparations belong also the various zwiebacks, made of prepared flours, such as, for instance, Opel's nutritious zwieback, Rademann's nutritious toast, Strohschein's beef-cakes, aleuronatzwieback, etc.

[The Health Food Co. furnishes good preparations.]

3. *Preparations Containing Fats.*

Lipanin contains 6 per cent. oleic acid, [Lipogen Chocolate].

v. Mering's Kraftschokodale [Hauswaldt Vigor Chocolate], (99.1 per cent. dry substance) contains 4.4 per cent. albumen and alkaloids, 20.97 per cent. fat, 72.44 per cent. anitrogenous substances, 1.25 per cent. ash (very palatable and well borne).

[A number of oil and egg emulsions are on the market.]

Extrabuccal Nutrition.

When for any reason whatever nutrition by the mouth is impossible, or when the stomach and intestines are capable of accepting and digesting only small quantities of food, or finally when for therapeutic reasons a temporary relief of the alimentary apparatus is indicated, the extrabuccal nutrition may form an important aiding measure to the therapy of nutrition. Three ways are open for the extrabuccal nutrition: (1) nutrition by the rectum, (2) subcutaneous nutrition, (3) nutrition through a gastric or duodenal fistula. Gavage may also be called extrabuccal to a certain extent.

1. *Nutrition by the Rectum* (*Nutrient Enema*).

The physiologic basis of the clysmata nutrientia, recommended and employed already by Celsus, had not been created before the investigations of Voit and Bauer,[32] as well as of Eichhorst.[33] The first-mentioned authors have established by animal experiments that proteids may be partly absorbed even without previous peptonization, but that common egg-albumen is resorbable only on addition of chloride of sodium.

Eichhorst found that also the proteids of the milk, as well as solutions of myosin and albuminates of alkalies, may be absorbed from the rectal mucous membrane. Experiments on the *human being* made by Czerny and Latschenberger[34] showed the same results for albuminates, and demonstrated furthermore that emulsified fat and starch-paste also are absorbed, whereby it remained in doubt whether the latter was saccharified before the absorption or whether it was absorbed as such.

1. v. Leube[35] recommended meat-pancreas clysmata as very useful and easily absorbable, and demonstrated this good assimilation by estimation of the urea.

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[32] Voit and Bauer, Zeitschrift für Biologie, 1869, Bd. 5.

[33] Eichhorst, Pflüger's Archiv, 1871, Bd. 4.

[34] Czerny and Latschenberger, Virchow's Archiv, 1874, Bd. 59, S. 661.

[35] v. Leube, Deutsches Archiv für klinische Medicin, 1872, Bd. 10, S. 13.

It is a very much mooted question how large the quantity of albumen and the amount of anitrogenous substances must be for maintaining the nitrogen equilibrium, and whether we actually may succeed in nourishing patients through exclusive or predominant rectal enemata. Voit and Bauer are of the opinion that only one-fourth of the proteids are absorbed and that it is impossible to nourish completely man or animal by the rectum, particularly as it is very difficult to introduce into the body the necessary quantities of anitrogenous substances.[36] A furtherance in the explanation of the question of the nutritive value of the nutrient enemata is due to Ewald[37] and Huber.[38] The former has demonstrated by estimation of nitrogen in the urine and fæces that egg enemata, prepared or unprepared, are absorbed just as promptly as the commercial albumoses, and that they may bring about a considerable increase in body weight, corresponding to that caused by peptones. Furthermore, Ewald has proved that albumoses do not offer better conditions for their absorption than common emulsified egg-albumen does. Huber, relying also upon the estimation of nitrogen, has substantiated the statements of the former investigators, and especially those of Ewald, and confirms the fact that egg-albumen is absorbed from the rectum, but simultaneously he showed that the addition of a small quantity of table salt (one gramme NaCl to one egg) favourably influences the absorption of emulsified eggs. The capability of absorption of the clysmata was not essentially increased by peptonising the egg.

This view, however, is not generally adopted. v. Leubé,[39] for instance, prefers the peptones, because, being absorbed more rapidly, they are less subject to putrefaction before absorption than the native proteids introduced into the rectum. According to Plantenga's[40] investigations, somatose, among the proteid substances, is most easily resorbed. To be sure, not more than 20 per cent. solutions must be employed, as otherwise diarrhœa occurs. Still, under the most favourable conditions, not more than 25 grammes can be assimilated, which would amount to 100 calories.

As to *carbohydrates,* it is known that starch (after previous saccharification), as well as the sugars, are absorbed from the mucous membrane of the rectum and colon. Hereby we must remember that highly concentrated solutions of sugar, irritating the rectum rather easily, are quickly expelled.

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[36] Bauer, v. Ziemssen's Handbuch der allgemeinen Therapie, 1883, Bd. 1, Thiel 1, S. 264.

[37] Ewald, Zeitschrift für klinische Medicin, 1887, Bd. 12, S. 407-425; Du Bois-Reymond's Archiv für Anatomie und Physiologie, 1899, S. 160.

[38] Huber, Korrespondenzblatt für Schweizer Aerzte, 15 November, 1890; Deutsches Archiv für klinische Medicin, 1891, Bd. 47, S. 495.

[39] v. Leube, Ueber extrabuccale Ernährung, Deutsche klinik am Eingang des XX Jahrhunderts, 1901, 1. Lieferung.

[40] Plantenga, Inaug.-Diss., 1898.

According to Plantenga, cane-sugar was absorbed the easiest, while grape-sugar very easily produced diarrhœa. According to v. Leube, higher concentrations than of 5 to 6 per cent. sugar must not be used. Starch solutions may be employed in solutions of 20 to 30 per cent., so that in an enema of 300 grammes, about 200 calories are introduced into the body.

According to the congruent investigations of Deuscher,[41] Plantenga, Stüwe,[42] and Strauss,[43] only 10 grammes of *fat* are absorbed. The absorption is increased by addition of pancreas in substance (v. Leube), or through previous emulsifying of the fat. According to E. W. Baum,[44] the absorption of fat is hereby considerably facilitated.

Technique and Composition of Nutrient Enemata.—Ewald[45] gives the following directions: 40 grammes wheat flour are mixed with 150 cubic centimetres of lukewarm water or milk, and 1 or 2 eggs with about 3 grammes of table salt and 50 to 100 cubic centimetres of a 15 to 20 per cent. grape-sugar solution are added and the whole mass is well stirred. The addition of a small quantity of alcohol, in the form of a glass of red wine, serves as a stimulant. The nutritive value of the mixture amounts to about 450 calories.

A cleansing enema of one-fourth litre of water or salt solution must precede the injection of the nutrient enema, and we must wait till the evacuations (sometimes several) have occurred; the quantity of the enema must not amount to more than 200 or 300 cubic centimetres.

The introduction is done either with a syringe attached to a long, soft-rubber tube or with the irrigator (fountain syringe), which may also be supplied with a soft-rubber tube with a wide opening. After the introduction the patient should remain for a few minutes in a recumbent or lateral position.

In addition to the eggs, I have for several years employed milk for nutrient enema, and I convinced myself that the greater part of the same disappears by absorption, while the smaller part curdles, evidently through bacterial action, and is expelled later. The enema recommended by me has the following composition:—

250 grammes milk,
2 yolks of eggs,
3 grammes of table salt,
1 tablespoon of red wine,
1 teaspoonful of wheat starch.

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[41] Deuscher, Deutsches Archiv für klinische Medicin, 1897, Bd. 58.

[42] Stüwe, Berliner klinische Wochenschrift, 1896, No. 20.

[43] Strauss, Charité-Annalen, 1897, 22 Jahrgang.

[44] E. W. Baum, Therapie der Gegenwart, September, 1902.

[45] Ewald, Therapie der Gegenwart, 1900, S. 433.

In a large number of *respective* (see below) cases I could convince myself that this quantity was readily absorbed by the rectum and that a small fraction of it was expelled only after many hours. Riegel[46] employs almost the same composition.

I apply the injection by means of a Hegar funnel and a long, soft rectal tube. The irrigator is less useful, because the latter must be removed again after the application of the cleansing enema; while in the use of the funnel, by simply lowering and elevating it the water may return from the rectum, and the nutrient enema may immediately be injected.

A considerable large amount of nutriments may be brought to absorption, if, as I have practised it for several years, the fluid is allowed to flow drop by drop into the intestine, which is done preferably by supplying the afferent tube with an appropriate clamp. Under these circumstances we may succeed, though not always, in introducing about 1 litre of milk a day, which amounts to 600 calories. The single doses of the injected fluid, when applied in this manner, may amount to 500 cubic centimetres, and the procedure may be repeated at least three times a day. Thirty to forty minutes are spent in the flow of the nutritive fluid.

2. *Subcutaneous Nutrition.*

In this method of nutrition, neither proteids in their native form nor in the form of albumoses or peptones come under consideration, because the former remain unchanged after subcutaneous introduction and are eliminated in the urine, while albumoses are not assimilated and in addition produce toxic effects. There remain, therefore, only the fats and carbohydrates for subcutaneous introduction.

As to carbohydrates, F. Voit,[47] to be sure, has demonstrated that considerable quantities of sugar (up to 60 grammes and more) can be taken up through the skin into the blood. On the other hand, as experience shows, the subcutaneous application of sugar meets with great difficulties in the practice, on account of its painfulness—v. Leube observed fever at the application, and warns directly against further experiments.

Of greater prospects appears to be the subcutaneous fat-nutrition. v. Leube[48] deserves great credit for having introduced it into practice. According to this author, 50 to 100 grammes of oil are readily injected under the skin, preferably by allowing the oil, which was sterilised by boiling, to flow slowly from a funnel closed with cotton, or by its being pressed under the skin by means of a special pressure apparatus (du Mesnil, Ewald, Sahli, Strauss). Unfortunately, this procedure is not entirely painless. According to Ewald,[49] it cannot be done for any length of time in sensitive patients. The method, therefore, has not been generally adopted.

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[46] Die Erkrankungen des Magens, Wien, 1896, S. 247.

[47] F. Voit, Münchener medicin. Wochenschrift, 1896, No. 31.

[48] v. Leube, Verhaundlungen des 13. Krongresses für innere Medicin, 1905.

3. *Nutrition Through Gastric and Jejunal Fistulæ.*

This form of nutrition comes under consideration either when the entrance to the stomach is permanently occluded, or when the stomach has become incapacitated by corrosive poisons or carcinomatous growth to exercise its digestive function.

In the former case nutrition is administered through a gastric fistula, in the latter case (according to Maydl's procedure), through a jejunal fistula.

If there is a prospect of gradually removing the obstacle in the passage (as, for instance, in cicatrized strictures of the œsophagus), the nutrition through a gastric fistula is undoubtedly a most useful, indeed a life-saving measure, while in malignant processes of the œsophagus or the stomach, feeding *per fistulam* can, of course, be expected only to postpone the unavoidable end for a short time.

4. *Gavage, Alimentation Forcée, Suralimentation* (*Debove*[50]).

Gavage also belongs, to a certain extent, to the extrabuccal feeding. Debove was the first to make the attempt of curing tuberculosis of the lungs by means of pouring strengthening aliments (milk, eggs, meat-powder) into the stomach, and others, particularly French physicians (above all Dujardin-Beaumetz), followed him in this method. Peiper[51] and later principally v. Leyden,[52] pointed to the excellent effect of gavage in certain cases. In diseases of the digestive apparatus, the same has not been tried, as far as I know, but in my opinion it is of a certain importance also in this respect. According to my experience, based, to be sure, only upon a few observations, gavage comes under consideration with a view of success in all those cases in which appetite has been reduced so much that there is danger for the integrity of the organism. For instance, I saw some cases in which anorexia was the only one and essential symptom of the disease, and in its existence continuing for months and years there was not an inconsiderable danger for the organism. Although the *indicatio causalis* is not fulfilled hereby, there is in the forced alimentation a safe and simultaneously harmless remedy against the loss of strength, which under all circumstances should be tried.

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[49] Ewald, Therapie der Gegenwart, October, 1900.

[50] Debove, Du traitement de la phthisie pulmonaire par l'alimentation forcée, Paris, 1881.

[51] Peiper, Deutsches Archiv für klinische Medicin, 1885, Bd. 37.

[52] v. Leyden, Verhandlung des Vereins für innere Medicin, Jahrg. 8, S. 74.

*Indications for Rectal Feeding.**

If we take into consideration the affections of the œsophagus, which naturally are closely connected with gastric affections, we have to mention above all the cases with stenoses and strictures of the œsophagus, then the diverticles, as fit objects for the rectal feeding and, as a matter of course, only in that stage in which fluids cannot pass at all or only partly into the stomach.

Spasms of the œsophagus or cardia (œsophagism, cardiospasm) will only exceptionally and temporarily make rectal feeding necessary.

In the just-mentioned cases, rectal feeding corresponds exclusively to the *indicatio vitalis,* while in the following gastric affection a second indication is added: protection and relief or avoidance of the affected organ. Such is particularly the case in cancer of the stomach, benign stenosis of the pylorus or duodenum with consecutive ectasia and the gastric ulcer.

In *cancer* of the stomach, especially that of the pylorus, the maintenance of nutrition by rectal enemata is justified by the defective propulsion of the ingesta and its resulting developments of toxic products which unfavourably influence stomach as well as intestinal portions. But in gastric cancer rectal feeding must be employed only as a supplement to buccal feeding. For, as far as my experience goes, rapid debility ensues if rectal feeding is employed exclusively. Another reason for rectal feeding is the fact that the tissue loses its amount of water as a result of deficient resorption in the stomach, a condition which unfavourably influences the whole body.

The support of nutrition by means of alimentary enemata is plain and generally recommended by a majority of writers in cases of *benign stenosis of the pylorus.* In cases of this kind the danger for the organism rests principally in the defective resorption of fluid. A practical standard for the necessity of systematic introduction of fluid by the rectum is found in the quantity of the urine. If this is reduced substantially below 1000 cubic centimetres within twenty-four hours, I usually have rectal enemata administered. The favourable influence is manifested not only by the increased diuresis, but also by the decrease of the tormenting thirst, and also, though rarely, by increase of body weight. Plain water or water with table salt (Wegele[53]), one-half litre two or three times a day, may be injected, or, according to Fleiner's[54] advice, slightly salted beef broth, or a mixture of beef broth (two-thirds) and white (Rhine) wine (one-third) may be applied. Of course, the above-mentioned enemata, containing a certain nutritive value, are to be preferred to the injections of plain water.

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* As the subcutaneous feeding has not yet obtained a definite indicative basis on account of the above-mentioned difficulties, we will consider only the rectal feeding in the following.

In several particularly severe cases of pyloric stenosis which were characterised by excessive fermentative processes, I have, in order to remove the latter and simultaneously to give relief to the stomach, had *exclusive* rectal feeding administered for ten days to two weeks, and that with strikingly good result, although this was of course but transitory. This favourable influence was shown, apart from the better subjective well-being, through the disappearance of bacteria (yeast-fungi, bacilli of various kinds), through evidently better absorption (abundant diuresis, improvement of the bowel-movements), even through temporary gain in weight. In one of my observations this result continued for three or four months, a result not to be underrated compared with a gastric affection stubbornly defying all therapeutic measures (lavage included). Rössler[55] and Schlesinger[56] also followed a similar procedure, with favourable effect, in several cases of gastric dilatation. Gastric ulcer also forms a not less important indication for partial or complete rectal feeding. Hereby we have strictly to distinguish the ulcer at the stage of bleeding and shortly after, from the chronic ulcer during cicatrisation or the recrudescent ulcer.

At the stage of bleeding and several (*in maximo* five) days afterward, the *exclusive* rectal feeding, if feeding is at all necessary, is the best and safest method of treatment, because the preconditions for a rapid formation of thrombi to any satisfactory extent are fulfilled.[57] As, moreover, rectal feeding is entirely sufficient for the maintenance of the patient for the time being, a deviation from it, such as, perhaps, in form of synchronous oral feeding, is without reason.

Not earlier than three days after cessation of gastric hæmorrhage we may, besides the rectal feeding, cautiously begin in the usual manner (milk, beef-tea, bouillon, soft-boiled eggs, tea, etc.) with the ingestion of a liquid diet.

Besides, at the stage of hæmatemesis, attention has been called, especially by English physicians, among whom I will mention McCall Anderson[58] and particularly Donkin,[59] to the favourable results obtained by means of exclusive rectal feeding in cases of chronic recrudescent ulcers. Donkin extended the time of rectal feeding as long as twenty-three days in several cases, and reported excellent results. Indeed by these results, I was the first in Germany to employ exclusive rectal feeding *in cases of severe, frequently-recurrent gastric ulcers concomitant with severe gastralgia, in which the usual curative measures had had either no effect at all or but a transitory one.* The period of rectal feeding, during which, of course, the patient must be confined to absolute rest in bed, lasts usually ten days to two weeks. Three, rarely four, injections of the above-mentioned mixture are applied within twenty-four hours. No medical treatment (except hot Priessnitz's umschläge or hot poultices) is applied.

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[53] Wegele, Münchener medicin. Wochenschrift, 1894, No. 12.

[54] Fleiner, Lehrbuch der Krankheiten der Verdauungsorgane, 1896, 1 Hälfte, S. 367.

[55] Rössler, Wiener klin. Wochenschrift, 1893, No. 40.

[56] Schlesinger, *ibid.*, 1895, No. 19.

[57] Compare Special Part, page 455.

If thirst is great, we may allow the patient to drink small quantities of an alkaline acidulous water, but I try to avoid it if possible. After the end of the abstinence cure, I begin, after a few days, with strengthening but easily assimilable *solid* diet (meat, wheat bread, vegetable purées, farinaceous food), which then is frequently well borne without the occurrence of pain.

My experience about rectal feeding refers to more than a hundred cases, a third of which were observed in my clinics. I regard my judgment as fairly conclusive. I may state that in one part of the cases the rectal feeding is well tolerated, and that it removes pain almost *immediately* and *permanently;* in another part of equal number the result is good, though not permanent; if the natural feeding is resumed, pain recurs. There are other cases in which no effect at all is observed. It is, however, doubtful whether in these (rather rare) cases we had not to deal with gastric neuroses which may simulate occasionally an exact picture of ulcer. Finally, there are some observations of cases which taught me that occasionally the sensitiveness of the stomach made the application of nutrient enemata impossible.

As is readily seen, the result of the nutrient enemata, continued for some days, is not absolutely certain, nor is the method a pleasant one. Indeed in private practice, without sufficient trained attendance, the method is absolutely impracticable. I observed severe collapses, delirium, inanition, etc., in private practice, which compelled me to interrupt the procedure.* At any rate, the rectal feeding in cases of gastric ulcer should be only the *ultimum refugium* [if operation is denied], and only if the diagnosis is absolutely sure. If Ratjen,[60] in more recent time, recommends rectal feeding as the sovereign cure for ulcer, he misjudges the difficulties which are associated with a systematic rectal feeding, continuing for ten days to two weeks, for the patient as well as the physician and the nurse. Quite apart from this, good results are obtained by means of the usual and less severe methods in about 88 per cent. of the cases.

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* [The method ought not to be undertaken under any circumstances without the assistance of an intelligent trained nurse.]

[58] McCall Anderson, British Medical Journal, 10 May, 1890.

[59] H. B. Donkin, The Lancet, September 27, 1890.

An important indication for rectal feeding is finally given by the frequent cases of nervous and uncontrollable vomiting or hyperæsthesia of the stomach (irritable stomach); the partial undertaking of the feeding by the intestine is indicated not only by the grave debility of the whole body, but rather more so because the relief of the stomach undoubtedly soothes the gastric nerves.

Appendix.

Dietetic Cures in Gastric Diseases.

1. *Milk Cures.*—In the therapeutics of digestive disturbances milk is of essential importance; it is used against the most various affections of the gastro-enteric canal, frequently with excellent result, sometimes with little effect, not seldom with great injury to the patient. This diversity of its effect is brought about not only through the above-mentioned varied tolerance toward this food, but also through the peculiar changes to which milk is subjected in the alimentary canal and which at the present are only slightly known to us.

In the meantime, however, we possess at least a few clinically useful hints, valuable for the digestion of milk and its influences upon the metabolism, which are adapted to guide the administration of milk-cures out of the wide sea of empyricism into the narrow channel of scientific criticism.

In discussing this question we have strictly to distinguish between systematic milk-cures as originally carried out and established by Karell, and the occasional addition of larger or smaller amounts of milk to the remaining food-portion.

As to the former, we must never forget that milk, even in its largest quantities (about 3 litres a day), does not suffice at all to cover the need of nitrogen, and particularly that of carbohydrates and fat. It is true this only holds good for the healthy individual under normal conditions, and it is undoubted that the functionally *weakened* organism may, as the metabolic investigations of Fr. Müller[61] and Klemperer[62] have taught, be put into equilibrium with this number of calories; we must, however, not forget that in cases of this kind the power of assimilation of the organism is greatly reduced and the alimentary canal is not adapted for the resorption of large quantities of food-substances. If at all, it is especially necessary in intestinal affections to combine great nutritive value with small substance, that is, to administer the nourishing food in condensed form.

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[60] Ratjen, Deutsche medicinische Wochenschrift, 1896, No. 52.

[61] Fr. Müller, Zeitschrift für klin. Medicin, 1889, Bd. 16, S. 496.

[62] Klemperer, *ibid.*, S. 550.

On the other hand, it must be admitted that in some cases of digestive affections, milk forms not only a sovereign nutriment, but a decided remedy. Such is particularly the case in follicular gastritis and in ulcer of the stomach or duodenum. In such cases where, especially in recent cases, it is necessary to avoid all mechanical irritations, and furthermore to bind the excess of free acidity, the milk-cure fills, as we have known for a long time, its place better than any other food. [In recent time, Lenhartz[63] is rather averse to the recommendation of milk in gastric ulcer.]

As far as my observations allow me to judge, milk-cures are also useful in those forms of dyspepsia which manifest themselves usually in complication with other diseases (phthisis, anæmia, etc.) "under the clinical picture of gastric insufficiency." In cases of this kind milk is well tolerated and well assimilated. Also in a few cases of nervous dyspepsia, when objective disturbances are absent, milk-cures may be followed by results, but by no means in all cases; there is no rule. Therefore, I begin with a kind of preliminary treatment before employing a systematic milk-cure; its effect will be decisive for the actual "cure."

Milk is frequently very badly borne in dyspepsias with constant loss of HCl; the removal of the yellow, thick-fluid, uncurdled masses with the strong odour of fatty acids from the stomach will readily give a conception of the complaints of these patients after ingestion of milk. The deficiency of coagulation is combined with the danger of abnormal decomposition in the alimentary canal. On the other hand, I observed recently and frequently favourable effects of so-called "*pegnin*-milk" (milk finely coagulated by lab-ferment) in cases of inacid gastritis.

Milk has an important place in all obstructions of the pylorus, though not in form of milk-cures in the meaning of Karell, but as a partial feeding —in repeated small doses, cooled, carefully sterilised, and, if necessary, boiled with various flours (page 298). Milk is almost indispensable when motor insufficiency is combined with chemical insufficiency, namely, in chronic pyloric hypertrophy or pyloric carcinoma, etc. Only fluid substances can pass the stenotic pylorus. In a case of chronic pyloric hypertrophy I succeeded by means of exclusive feeding with fluids (4 to 5 litres of milk within twenty-four hours), in preventing the formation of stagnation in the stomach, in increasing the diuresis to 2 litres and in obtaining considerable increase of weight of the exceedingly emaciated patient.

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[63] Lenhartz, Medical Record, June 16, 1906.

Milk-cures in the strict sense are less adapted in atonies and mechanical insufficiency without stenosis. In such cases large quantities of milk would counteract the indication for the best possible protection of the gastric musculature. Experience also speaks against the use of systematic milk-cures in these cases. The systematic use of milk seems to me also contra-indicated in cases of severe flatulency, in chronic diarrhœa, in intestinal tuberculosis, amyloid degeneration of the intestine, as well as in ulcerative processes of the colon associated with diarrhœa. There are, however, some cases in which milk, and particularly hot milk, is well borne.

In many cases of milk-cures, the intestines have more to be looked after than the stomach. In a great number of digestive affections there exists, for instance, constipation as a very troublesome symptom which is occasionally increased by the systematic ingestion of milk.

The digestibility of milk may be more or less increased by certain admixtures, such as barley or farina decoctions (1 part milk to 2 or 3 parts of decoction), or also by the addition of lime-water or lime-powder, and finally by the addition of cognac or arrac (10 to 200); the addition of alcohol, particularly, perhaps on account of its bactericide action, frequently effects better conditions for digestion.

A very useful milk preparation in some cases of dyspepsia is the *cream*, on account of its extraordinary amount of fat (14 to 20 per cent.). Its administration is especially indicated when larger quantities of fluid have to be avoided; to be sure, subjective tolerance must not be forgotten. Similar to cream is the "Kraftmilch," suggested and recommended by W. Jaworski;[64] it also is distinguished for its high amount of fat. Jaworski used two kinds, one with 10 per cent. of fat, 1.8 per cent. of proteids, and 6 per cent. of milk-sugar, and another with 7 per cent. of fat, 1.8 per cent. of proteids, and 6 per cent. of milk-sugar. The manufacturing of the milk is done in such a manner that fresh milk is first diluted with water, the cream of 10 per cent. fat is taken by centrifugation, and 6 per cent. of milk-sugar is added.

Attention has been called to Gärtner's *fat-milk*, which is produced in such a manner that fresh cow's milk is diluted with water till the amount of casein is equal to that of mother's milk. The mixture is then centrifugated so far that the milk flowing out of the cream-tube possesses the amount of fat of the mother's milk. The deficit of sugar is replaced by a corresponding quantity of sugar of milk. Hereby a milk is obtained which resembles mother's milk as to its essential ingredients. Correspondingly, Gärtner's fat-milk coagulates also on artificial digestion in finer floccules than cows' milk; and, according to recent investigations by Emil Schütz,[65] Gärtner's fat-milk leaves the stomach quicker than cow's milk. According to my experience, which conforms very closely to that of Riegel,[66] the same is much better tolerated by the majority of stomach-patients than cow's milk. Other milk preparations are *concentrated* milk, *condensed* milk, *evaporated* milk, and various *milk powders*. Vegetable milk did not find a *general* adoption for dietetic purposes in gastric diseases.

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[64] W. Jaworski, Therapeutische Monatshefte, 1897, Maiheft.

[65] Emil Schütz, Wiener klin. Wochenschrift, 1896, No. 48.

[66] Riegel, Die Erkrankungen des Magens, 1897, S. 225.

Another dietetic food, frequently used in gastro-enteric diseases since Krukenberg's recommendation, is *buttermilk*. It is frequently administered, partly on account of its pleasant and refreshing taste, partly on account of its influence (due to its amount of lactic and butyric acid) upon peristalsis. Similarly to buttermilk, *sour milk, sour cream, koumys,* and *kefyr** are used. I frequently observed satisfactory results from the "two-days" *kefyr* in cases of intestinal atony, and generally I have employed the "three-days" *kefyr* in habitual diarrhœas, usually with excellent effect. ["One-day" kefyr has been recommended as an excellent remedy in cases of constipation.]** Only occasionally it is not well tolerated, causes abnormal fermentation, and therefore has to be omitted. The efficiency of kefyr has also been proved through the publications of Weiss,[67] Eckervogt,[68] Podwyssotzki,[69] and others.

2. *Weir Mitchell Cure*[70] (*Playfair Cure,*[71] *Feed and Rest Cure*).—In gastric neuroses, as in other neuroses, attempt has been made to ameliorate the nutrition and to strengthen the general state of health by the use of the so-called feed and rest cures. On the whole, however, the experiences of the authors are but slightly encouraging as to nervous dyspepsia. According to Burkhardt, the Mitchell cure has no positive effect in nervous dyspepsia, and frequently even an unfavourable influence. Furthermore, according to the author, some of the indications against the feed and rest cure are as follows: irritative conditions of the brain, both depressive as well as excitative; then hysteria with uncontrollable vomiting and visceral neuralgias, which are recognised as affections of the sympathetic nerve. Specifically adapted for the Mitchell cure may be considered those types of neurasthenia and hysteria in which a high degree of anorexia and consecutive great emaciation, but otherwise objectively good digestion, are present.

According to my numerous observations, those cases are most adapted for a feed and rest cure, in which a condition of sub-nutrition exists along with normal gastric and intestinal functions. Indeed, the nervous dyspepsia, as far as it is associated with sub-nutrition, yields, as I will emphasise in contrast to Burkhardt, the most favourable prospects. In these cases good results are always obtained. Different, however, are the cases in which nervous dyspepsia is complicated with a true organic affection, namely, gastritis, myasthenia, ectasia, etc. In cases of this kind I have frequently observed acute digestive troubles (vomiting, diarrhœa, pressure, pain), which demand an interruption of the cure.

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* [Kefyr can readily be made by the new method of employing the kefyr tablets.]

** [Kefyr has been introduced into this country only a short time; good kefyr may be had in Philadelphia and other cities.]

[67] Weiss, Klin. Zeit- und Streitfragen, Wien, 1890, Heft 10.

[68] Eckervogt, Kefir, seine Darstellung aus Kuhmilch, Berlin and Neuwied, 1890.

[69] Podwyssotzki, Zeitschrift für diätet. und physikal Therapie, 1902, Bd. 5.

[70] Weir Mitchell, Fat and Blood, third edition, 1884, and Die Behandlung gewisser Formen von Neurasthenie und Hysterie, übersetzt von Dr. G. Klemperer, Berlin, 1887.

[71] Playfair, Systematische Behandlung der Hysterie und Nervosität, Deutsch von Tischler, 1883.

As the number of feed-rest-cures undertaken by me in cases of nervous dyspepsia in the course of the past fifteen years is rather large, I ask leave to give a few rules for its technique, without detailing too much. The patients have to keep absolute rest in bed for four to five weeks. Nourishment is given every two or three hours and is to be made full of changes. The large quantities of milk which originally were recommended as an integral part of the cure are, as far as my experience goes, not all necessary; on the contrary, they are, in cases of gastric atony, a direct burden. They are replaced preferably by half to one litre of cream in doses of 150 to 200 cubic centimetres each. The diet ought to contain plenty of carbohydrates and fats, but under no circumstances "Genussmittel" (refreshments, relishes) must be left out of consideration. When constipation exists, we have to take notice of it in the diet by addition of plenty of sweets (sugar of milk, mannit, honey, marmalade, preserves), as well as acids (buttermilk, sour milk, kefyr, cider, Moselle wine, lemonade, etc.). Massage and faradisation act, in my experience, essentially by suggestion. The result, that is, the subjective well-being and the increase in weight, is hardly influenced by them. On the other hand, massage is perhaps of value in the better distribution of fat,* which in women may be of cosmetic importance.

For better understanding, I give herewith the directions which have proved efficient in numerous cases:—

7 o'clock:—

¼ litre Vigor chocolate in cream.
3 to 4 zwieback (2 rolls).
20 to 30 grammes butter.

9:30 o'clock:—

Cold or warm meat, eggs, egg-foods, light or white bread (perhaps Graham bread), butter 20 grammes.
150 grammes cream.
Preserves, sweet (stewed fruit).
Farinaceous foods.
1 to 2 glasses of cider or other fruit-wines (perhaps also some raw fruit), [grape juice].

12 o'clock:—

150 grammes cream.
2 or 3 biscuits (crackers).

2 o'clock:—

¼ litre soup.
$\left.\begin{array}{l}\text{Vegetables}\\ \text{Potatoes}\end{array}\right\}$ in purée form.
Meat and fish.
Salad.
Stewed fruits (sweet).

4:30 o'clock:—

Coffee, tea with cream (150 grammes).
Zwieback, cakes, Graham bread, butter (20 grammes) or honey.

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*[According to more recent observations, massage will not reduce fat; on the contrary, it will rather increase the adipose tissue, which latter fact may be also of cosmetic as well as physical importance in very lean patients.]

8 o'clock:—
Cold or warm meat, or fish.
Eggs, egg-foods.
Light bread, Graham bread, butter (30 grammes).
Stewed fruit.
2 glasses of fruit-wine or 1 bottle of malt beer.

9:30 o'clock:—
200 grammes cream with 2 or 3 crackers.

Of course, this diet may be varied, indeed in some cases I revise the bill of fare from day to day, and make changes until the wishes of the patient coincide with my intention of increasing the weight. The increase of weight obtained by me under this regimen amounts on an average to six to eight kilos (twelve to sixteen pounds) within four weeks. We need, of course, not mention that there occur also disappointments in contrast to these favourable effects. In some cases, especially in continuation of a regularly undergone feed-rest-cure, I have the patients use "half feed-rest-cure" for one or two months. I mean by this a rest in bed till twelve o'clock noon, after which time the patients may move around in the room, or may take a walk for one or two hours. The same cure I use also in patients whose circumstances do not allow an exclusive rest-cure of several weeks' duration. The results of the latter are, of course, not to be compared with the regular rest-cure, but they are, nevertheless, rather satisfactory, provided the patients exhibit the necessary energy.

If the patients are not urgently directed to continue a mode of life analogous to the feed-cure for the following few months, they lose very often their hard-gained fat in a short time.

[That fresh air is a necessary aid in rest-cure demands no further explanation. Windows must be kept open day and night; and rooms where the noise of the streets cannot reach are to be preferred; a protected porch or sun-parlor is an excellent help in the undertaking of a rest-cure.]

3. *Vegetarian Diet.*—The vegetarian diet in its strictest sense, that is with exclusion of milk, butter, cheese, eggs, can hardly be considered at the present time from a scientific standpoint, because the same, in spite of a few exceptions, must be regarded essentially as a diet of abstinence. In connection, however, with the above-mentioned animal products, as lacto-vegetable diet (Albu), it is of great value in certain gastric and intestinal affections. To these belong, above all, those cases which, without exhibiting any anatomical lesions, present a certain relaxation of the alimentary canal (nervous gastric and intestinal atony) due to too bland a diet. For such cases the lacto-vegetable diet, as Albu[72] recently anew emphasised, acts, as a rule, excellently by its favourable influence upon the whole nervous system, and especially because, through regulation of digestion, it quickly removes numerous localised gastric and intestinal troubles. Whether the other animal substances, such as meat and fish, have to be banished entirely from the bill of fare depends upon the type and degree of the general and local

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[72] Albu, Die vegetarische Diät, Leipzig, 1902, George Thieme.

neurasthenia. I usually content myself with a restriction of the animal proteids in the majority of cases. Apart from this, the lacto-vegetable principle cannot always be carried out, particularly not for any length of time and with all its consequences. Frequently, patients have not the energy (sometimes they find it impossible) to continue a mode of life contrary to their accustomed social habits. Not incorrectly, Albu attributes a main part of the results of the lacto-vegetable diet to the abstinence from alcohol, which, in my experience, must always be most strictly carried out in neurasthenics, especially in the patients suffering from gastric neuroses.

4. *Grape Cures.*—In some cases of gastro-enteric diseases *grape-cures* have been successfully employed. Their principal effect depends evidently upon the large amount of sugar in the grapes (according to König, 14.36 per cent. on an average). Through this fact the diet acts in two directions: laxative and lowering the amount of acidity. In consequence Bialocour[73] recommends the grape-cure especially in superacidity, in which he observed temporary disappearance of hydrochloric acid secretion. Of undeniable usefulness is the grape-cure as an aid in habitual constipation. To be sure, according to my observations, its influence is not more lasting than the use of saline and Glauber salt waters. In all these cases, the *grape-juice* is to be employed without skins and seeds, because, according to Laquer,[74] the skins have a rather constipating effect, due to the amount of tannic acid contained in them.

Remarkable are the favourable results which Hausmann[75] in Meran (Tyrol) obtained by means of grape-cure in cases of *ptyalism* if the latter was connected with disturbances in the stomach and particularly in the intestines. Among the gastric affections adapted for grape-cures are, according to Hausmann, dyspepsia as it occurs in chlorotic and anæmic girls who are suffering from dysmenorrhœa, and dyspeptic conditions in women in so far as they are due to losses of blood or to nursing; even in advanced neurasthenics with dyspepsia, favourable curative results were observed by Hausmann.

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[73] Bialocour, Pamietnik Towarzystwa Lekarskiego, 1896, 9-34; quoted in Maly's Jahresbericht für Tierchemie, 1900, Bd. 30, P. 408.

[74] B. Laquer, Zeitschrift für diätetische und physikalische Therapie, 1899, Bd. 3, Heft 1.

[75] Hausmann, Therapeutische Monatshefte, September, 1887.

CHAPTER X.

Balneotherapy

Balneotherapy plays an important part in the treatment of digestive diseases. We have to deal here, as in other internal diseases, partly with the use of mineral waters (natural and artificial), partly with applications of baths of various types and composition, finally with climatic cures (mountain and sea-shore). They are often combined with various other therapeutic methods, such as massage, electricity, hydrotherapy, and gymnastic measures.

Every physician who has to decide on the selection of a health-resort knows that the balneotherapeutic ordination belongs to the most difficult tasks of the total therapy. For, while in the other branches of treatment we are usually capable of conforming to the supreme demand of the same, namely, the "nihil nocere," in the balneotherapeutic directions we move, though not always, at least frequently, on rather unsafe paths.

If this fact is valid for any branch of medicine, it is certainly so for the affections of the digestive organs. On the one hand we observe grand results under the use of mineral waters, which seem to put the medicinal treatment entirely into the shadow; on the other hand, disappointments, annoying equally to patients and physician, are not rare at all.

An essential drawback rests in the absence of precise indications, and especially of *contra-indications.* As usually the former are drawn in too wide limits, and the latter in too narrow limits, the physician, in selecting an appropriate watering place or health resort, stands often before an *"embarras de richesse,"* in which, if not possessed of personal experience, he can hardly finds his way. The resort physicians also would be better off if they would restrict as much as possible the limit of the indications for their resorts.

In recent years the study of the gastric functions, and especially the examination of the metabolism under the influence of mineral waters, has swept away many traditional mistakes, and among the many services of v. Noorden and his disciples, Dapper, Fr. Krauss, and others, there is particularly that of having applied the axe of experimental criticism to the mysticism of balneology, as it was rooted for centuries. Although this, according to my impression, may have been done a little too radically, and although I am not of the opinion that everything that has existed in balneology up to the present time is worthy of perishing, the attempt itself of bringing light out of the chaos of obscure doctrines and ideas deserves general recognition. As a fundamental gain of the researches of v. Noorden, Dapper, and Ludwig, the dictum may be put forth that *neither the Carlsbad water nor the saline waters influence the metabolism in any recognisable manner.* The hitherto frequently heard and thoughtlessly repeated words that this or that mineral water* has an ameliorating influence upon the metabolism, are accordingly to be put into the sphere of fables.

Through Brandenburg's[1] investigations it was established that, as was not otherwise expected, the elimination of fat and nitrogen was augmented during the use of Carlsbad cure. On the other hand, Ludwig could prove that the use of Carlsbad water does not essentially influence the N-equilibrium in spite of moderate diarrhœa. The phosphorus equilibrium, however, was negative during the drinking period; the loss of phosphorus must be ascribed to the loss of nuclein in the fæcal evacuations; in consequence of it the elimination of alloxur-nitrogen in the urine was also diminished.

In the following I am recording that which has proved itself useful to others and myself in the close observation of balneotherapy as to the treatment of digestive disturbances.

If we consider the *mineral* waters, there are particularly seven groups to be mentioned: —

1. Alkaline acidulous waters. Chief ingredients: sodium bicarbonate, carbonic acid.
2. Alkaline-muriatic acidulous waters. Chief ingredients: sodium carbonate, sodium chloride, carbonic acid.
3. Glauber salt waters. Chief ingredient: sodium sulphate.
4. Sodium chloride waters. Chief ingredient: sodium chloride (common salt).
5. Iron waters [chalybeates]. Chief ingredients: iron oxide, bicarbonate, or sulphate.
6. Lime waters. Chief ingredients: calcium carbonate, magnesia carbonate, or calcium sulphate.
7. Bitter waters. Chief ingredients: sodium sulphate and magnesia sulphate.

1. *Alkaline Acidulous Waters.*—Their action can be attributed to the amount of sodium bicarbonate and carbonic acid.

The first-named ingredient gives the alkaline acidulous water a prominent part in all gastric affections associated with an excess of hydrochloric acid. To these belong the common types of hyperchlorhydria, continuous gastro-chylorrhœa, pyrosis hydrochlorica. The action may be explained in such manner that a part of the excessive free HCl is neutralised, or through the continuous use of sodium bicarbonate the conditions for the HCl secretion in the blood may be so changed that a less acid gastric juice is secreted.

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* It is not probable that other mineral waters behave essentially differently from the mentioned waters, although experimental proofs are missing.

[1] Brandenburg, Therapeutische Monatshefte, December, 1899.

There is still a wide disagreement whether through the use of the said waters an increased bile secretion and—as I showed on human beings—a concomitant increased pancreas secretion are resulting, through which the duodenal digestion is favourably influenced. Nasse, and after him Röhrig, found that sodium carbonate diminishes the hepatic action; Rutherford and Vignal did not observe any influences at all, and only Lewaschew, who made the most thorough investigations of this subject, found increase of bile secretion. Likewise, a weak cholagogue action is ascribed to sodium bicarbonate by Rutherford and Vignal, a slight action by Prevost and Binet, a considerable action by Lewaschew, and no action whatever by Stadelmann.[2] At any rate, the action of alkaline acidulous waters upon bile secretion is at least doubtful.

The same may also be said of the influence of alkaline acidulous waters upon gastric secretion. Wendriner, at least, could *not observe any* change of the same, although artificially heated Neuenahr Sprudel was used in numerous cases. He found, however, as Ewald and Sandberg,[3] Spitzer,[4] Latkowski[5] have proved in using Carlsbad and Marienbad water, that the motor function was increased at least as long as the water was administered. But it is questionable, and not established through other investigations, whether this stimulation of the motor function is lasting. This would be the salient point for the favourable influence. According to (not published) investigations of my own, this is to be denied, at least in regard to Carlsbad water.

We know little about the importance of *carbon dioxide* in these and similar waters. For a long time an influence upon the peristalsis has been ascribed to carbonic acid gas, but this view has no sure experimental foundation. It is sure that this influence, as the daily experience with carbonated beverages shows, cannot be considerable, but may perhaps be manifested in cases where there already exists an increased peristalsis. As far as I know, the only investigations in regard to the influence of carbonic acid gas upon the gastric are those of Jaworski[6] and Penzoldt.[7] The former found that after introduction of carbonic acid into the stomach the acidity of the gastric juice increased in two cases, but in a third case it did not do so. Further, Jaworski observed improvement of appetite after introduction of carbonic acid into the stomach. Penzoldt also could observe an improvement of the gastric digestion after use of carbonated water, and that in such a manner that the hydrochloric acid secretion starts earlier and on an average reaches higher degrees, and that, further, the ingesta remain in the stomach for a shorter time.

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[2] Stadelmann, Berliner klinische Wochenschrift, 1896, No. 9 u. 10.

[3] Ewald u. Sandberg, Centralbl. für die med. Wissenschaften, 1888, No. 16.

[4] Spitzer, Therapeutische Monatshefte, 1894, Aprilhefte.

[5] Latkowski, Wiener klinische Wochenschrift, 1899, No. 26.

[6] Jaworski, Zeitschrift für Biologie, 1884, Bd. 20.

[7] Penzoldt, Deutsches Archiv für klinische Medicin, 1902, Bd. 73.

Not the least beneficial action of carbonic acid seems to consist in the fact that eructation is promoted, and thereby injurious gases which develop in the course of digestion are expelled.

One side of the pharmaco-dynamic action of the alkaline acidulous water is looked for in the *mucus-dissolving properties;* but this action also is more theoretically supposed than practically proved. Besides, it is necessary not only to dissolve the mucus adhering to the gastric walls, but to remove the conditions for its formation.

Without doubt one of the therapeutic agents depends also upon the *temperature* of the water, which may be native or may be effected artificially. This action, which also plays an important part in the thermal waters of Carlsbad, is particularly, as to sensitiveness, of a practical importance not to be underrated. The irritability of the stomach, the great sensitiveness to difference in temperature and to stronger stimulation, though physiologic in itself, and above all the abnormal reaction upon many otherwise easily assimilable ingesta, are, as clinical observation teaches, reduced by means of thermal waters. In consequence the general health is ameliorated, appetite is improved, and the weight of the body increased.

In the following, we give a review of the most frequently employed alkaline acidulous waters, in the order of the amount of the carbonates of soda, whereby we may remark that the waters of Bilin, Vals, Vichy, and Fachingen, on account of the large amount of sodium bicarbonate and free carbonic acid, are best adapted for our purposes. The figures of the table at the head of page 318, as well as of other tables, are taken with slight changes from the excellent "Grundriss der klinischen Balneotherapie" by Kisch.

2. *Alkaline-muriatic acidulous waters* are waters which contain, besides carbonic acid gas and bicarbonate of soda, sodium chloride in prominent or at least effective quantity.

In these waters also, which represent a combination of three factors, a definite type of action cannot be stated. For, on the one side, the amount of sodium chloride (*vide* table) in the commonly used waters is so slight that we can hardly speak of an essential action; on the other hand, we can not look for a great therapeutic agent in the small amount of sodium bicar-

	Sodium Bicarbonate in 1000 cubic centimetres.	Free CO_2 in 1000 cubic centimetres.
Vals	7.18	1039.08
Bilin	4.60	1337.06
Fachingen	3.65	945.02
Vichy (Grande Grille*)	4.88	460.57
" (Hôpital*)	5.03	
" (Célestins*)	5.10	532
Fellathalquelle	4.20	609.12
Preblau	2.86	637.91
Salzbrunn	2.42	630.49
Geilnau	1.06	1468.80
Giesshübel	1.26	1537.70
Neuenahr*	1.05	498.50

bonate. As to the part which carbonic acid gas plays in such cases, we have above mentioned the corresponding investigations.

The *clinical* balneotherapy ascribes to the said springs an important position in the treatment of the chronic gastric catarrh, due particularly to atonic conditions. In my own experiences they are principally reserved for secondary forms of dyspepsia, especially those with incipient phthisis, emphysema of the lungs, bronchitis; further, for chronic enteritis, congestive liver, and abdominal plethora in milder degrees.

The most prominent alkaline-muriatic springs are compiled in the following table, according to the amount of sodium chloride and sodium bicarbonate contained in them:—

	Sodium Bicarbonate in 1000 cubic centimetres.	Sodium Chloride in 1000 cubic centimeters.	Free CO_2 in 1000 cubic centimetres
Sczawnicza (Magdalenenquelle)	8.45	4.61	711.5
Luhatschowitz (Vincenzbrunnen)	4.29	3 06	1452.6
Gleichenberg (Constantinquelle)	3.55	1.85	1149.7
Tönisstein	2.57	1.41	1269.6
Ems** (Fürstenbrunnen)	2.036	1.01	599.3
Selters	1.23	2.33	1204.3

We observe, in this table, at once the striking amount of sodium bicarbonate of Luhatschowitz (Moravia) and particularly of Sczawnicza (Galicia), which is associated with a considerable amount of sodium chloride and in the first named also with carbonic acid gas. Accordingly, the use of these springs could be most recommended in the above-mentioned alimentary disturbances.

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* Are thermal springs.

** Has thermal springs of 36° to 56° C.

3. *Alkaline-saline waters* (Glauber salt), distinguished by an important amount of sodium sulphate to which are added more or less large quantities of sodium bicarbonate, sodium chloride, and free carbonic acid. The springs are partly cold (Elster, Franzensbad, Marienbad, Rohitsch, Tarasp), partly thermal (Carlsbad, Bertrich).

The prototype of the said springs is represented by Carlsbad. As the curative effects of the Carlsbad thermal springs are well recognised, it is easy to explain that attempts were made to elucidate their pharmaco-dynamics, which up to the present time were clothed in misty darkness.

The credit of having made the first step in this direction belongs undeniably to Jaworski. He, on the one side, in a very circumstantial series of experiments, compiled that which was known already, and tested it in a critical manner; and on the other side he created new views for the explanation of the physiological action.

Jaworski's[8] observations yielded effects of the thermal waters upon (*a*) the gastric function, (*b*) the bile (and pancreas?) secretion, (*c*) the sensibility of the digestive apparatus.

Regarding the former, they effect a powerful stimulation upon the gastric mucosa, and that in such a manner that the *secretion of gastric juice is considerably increased. Repeated small doses act better than one large dose.*

A long-continued use of the Carlsbad thermal water (such as in a regular Carlsbad "cure") causes a decided *decrease of the acidity of the gastric juice.*

Jaworski gives the following resumé of his investigations: that very small quantities of Carlsbad water or salt are capable of stimulating the secretion of acid and digestive function; that larger quantities diminish gradually the capability of secretion in the stomach, so that secretion of acid and ferments does not occur any longer through the usual digestive stimulation. Carlsbad water acts upon bile secretion and duodenal function in such a manner that the former increases and the peristalsis of the duodenum is energetically stimulated. Finally, Carlsbad water acts also by carrying mucus collected in the stomach and recurrent bile into the intestines.

The diminution of the irritable condition is also not to be underrated as an effect of Carlsbad water, wherein, as already stated, the temperature plays an important part.

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[8] Jaworski, Deutsches Archiv für klin. Medicin, 1885, Bd. 37, Separatabdruk, Wiener medicinische Presse, 1888, No. 3 u. 4.

A certain restriction was given to these results by the investigations of Ewald and Sandberg,[9] who could not observe any noteworthy influences upon the acidity, peptonizing effect, or finally upon the lab-ferment. They agreed with Jaworski's results only in the fact that in persons with superacidity a decrease of the same occurred after the termination of the experiments (thirty to thirty-six days). Strauss,[10] however, could not convince himself of a regular change of the hydrochloric acid secretion. Indeed, Simon[11] observed that Glauber salt solutions increase the hydrochloric acid secretion in cases of subacidity, a statement which was disputed by Gintl.[12]

Regarding the influence of the Glauber salt waters upon the motility, Spitzer[13] was the first to call attention to their favourable influence. Latkowski,[14] on examination of the Marienbad water, could observe an increase of the acid secretion as well as of the motor function of the stomach. On the other hand, in Brandenburg's[15] experiments a regulated condition as to the acceleration of the gastric motility (in the normal stomach) was not found in the majority of the cases. But suppose that an increase of the gastric motor function exists, the already-mentioned objections (page 316) hold good here also. As we may see from these experiments, and in spite of many endeavours, the salient point in judging Glauber salt waters depends still essentially upon our clinical experience. We may derive from it about the following principles:—

The Carlsbad thermal waters are indicated:—

1. In recent cases of dyspepsia, particularly in those associated with superacidity and moderate constipation.

2. In gastritis acida, especially those with abnormal mucous productions.

3. In some (not nervous) forms of superacidity, continuous flow of gastric juice, pyrosis hydrochlorica.

4. In *milder* cases of atony of the gastric muscles, due to sedentary life, one-sided nourishment (soups), habitual constipation, and consecutive superacidity.

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[9] Ewald u. Sandberg, Centralbl. f. d. med. Wissensch., 1888, No. 16 u. 18.

[10] Strauss, Deutsche Medicinal-Zeitung, 1889, No. 37 u. 38.

[11] Simon, Zeitschrift für klin. Medicin, 1898, Bd. 35.

[12] Gintl, Verhandlungen des 17 Kongresses für innere Medicin, 1899, S. 345.

[13] Spitzer, Therapeutische Monatshefte, April, 1894.

[14] Latkowski, *l.c.*

[15] Brandenburg, Therapeutische Monatshefte, December, 1889.

5. In cases of insufficiency of the chemism and decrease (not loss) of free hydrochloric acid.

6. *As after-cure after healing of a chronic gastric and duodenal ulcer, particularly those forms associated with superacidity.*

7. In dyspepsia due to constipation and hepatic congestion, etc., as long as the primary affection has not reached extreme degrees.

The use of Carlsbad is contra-indicated:—

1. In advanced forms of dyspepsia, particularly those with loss of hydrochloric acid.

2. In all forms of *genuine* (proved by examination of gastric contents) chronic gastritis with loss of hydrochloric acid.

3. In gastric ectasia, whether due to advanced stages of atony or to pyloric stenosis.

4. In all forms of nervous dyspepsia, even if associated with well-preserved chemical and motor function.*

5. In all forms of dyspepsia associated with *stubborn* habitual constipation.

6. In suspected malignant processes of the alimentary tract.

In cases of 1, 2, 3, and 6, on an average, larger doses (500 to 600 cubic centimetres a day) of thermal waters, in the remaining cases only small doses (200 to 400 cubic centimetres) are allowed.

As regards to the various springs of Carlsbad, they are distinguished as well known, essentially through the temperature. As a whole, the medium warm springs, Felsenquelle (60.9°), Mühlbrunnen (57.8°), Schlossbrunnen (56.9°), Marktbrunnen (50°), Kaiserbrunnen (49.7°), are to be preferred to the springs of higher temperature, Sprudel (73.8°), Neubrunnen (63.4°), and Theresienbrunnen (61°). This is absolutely necessary, as v. Leube emphasises, in cases of gastric ulcer; on the other hand, in agreement with others I select preferably and with good effect the springs of high temperature in very small doses in catarrhal affections of the small intestine and colon.

An essential part of the excellent Carlsbad cure results, however, must be referred to the specific diet, which must be designated as "Schonungsdiät" ("sparing diet"). On account of this, there is a favourable effect in all those who indulge too much in eating and drinking and accordingly acquire a condition of gastro-enteric weakness. On the other hand, it has been proved through Ludwig's[16] and Friedr. Krauss's[17] examination of metabolism that, in spite of the use of Carlsbad water, an entirely normal absorption of fat may occur in the intestinal canal, an experience which has been known empirically for a long time to intelligent physicians. And indeed at the present time the formerly notorious Carlsbad "consumption" cures become more and more rare, in so far as fats are not any longer forbidden to such an extent as formerly.

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* In these cases, however, only the drinking is contra-indicated, not the sojourn in Carlsbad, which offers opportunities for the administration of hydropathic and gymnastic measures, massage, etc.

Among the other Glauber salt waters (*vide* table), several show a composition similar to Carlsbad, as, for instance, Rohitsch (Tempelbrunnen) and Franzensbad salt spring, while Marienbad and Elster deviate essentially from Carlsbad through almost a double quantity of Glauber salt, and Tarasp (Lucius Spring) through its considerable amount of alkalies.

I select the springs of Elster and Marienbad in those cases in which Carlsbad might be indicated, but in which a pronounced intestinal sluggishness exists; while I reserve Tarasp for cases in which an influence is desired not only upon the stomach, but also upon the nervous system, and also upon the intestines (habitual constipation), and for the reduction of excessive adipose tissue (increased bodily exercise, mountain tours).

The composition of the Glauber salt springs is shown in the following table, and that in order of the amount of Glauber salt:—

One Litre.	Sodium Sulphate.	Sodium Bicarbonate.	Sodium Chloride.	Free Carbonic Acid.
1. Elster (Salt Spring)	5.26	1.68	0.82	986.84
2. Marienbad (Ferdinand Spring)	5.05	1.82	2.00	1127.74
3. Franzensbad (Salt Spring) . .	2.80	0.96	1.14	831.42
4. Carlsbad	2.37	1.92	1.03	104.01
5. Tarasp (Lucius Spring) . . .	2.01	5.45	3.67	1060.00
6. Rohitsch (Temple Spring) . .	2.02	1.075	0.09	1129.02
7. Bertrich (to 32.7° C.)	0.90	0.26	0.435	120.09
		Magnesium Sulphate		
[8. Crab Orchard, Ky.	1.00	3.00]		

4. *Sodium Chloride Waters.*—Similarly to Glauber salt waters, there is also with the sodium chloride waters, an apparently unsolvable contradiction between theory and practice. Through experiments it was shown that sodium chloride influences unfavourably the gastric digestion, partly because it inhibits proteolysis, partly because it produces alkaline transu-

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[16] Ludwig, *l.c.*

[17] Fr. Krauss, Berliner klin. Wochenschrift, 1897, No. 21.

dation instead of gastric juice secretion, whereby this latter is made inactive through the former; practice, however, shows the contrary, and we must endeavour to reconcile these contrasts. According to my opinion, the mistake was made of starting from unequal premises when, for instance, Wolff,[18] with a view to the study of the influence of sodium chloride upon the gastric digestion, brings at one and the same time 5 grammes of table salt into the stomach; this dose corresponds to a litre of Rakoczy water (5 cups), a dose which will be hardly administered as a single dose.

There is no doubt in my mind but that small doses of table salt are of great balneodynamic effect, and I usually give preference to those among the sodium chloride waters which have a smaller amount of sodium chloride.

In some waters there is perhaps, beside the amount of sodium chloride, a considerable quantity of carbonic acid of importance. As the table on page 325 shows, Kissingen, Homburg, Pyrmont (Salzquelle), Soden (Milchbrunnen), [Kissingen Spring and Congress Spring, Saratoga, N. Y., Hathorn Spring, Saratoga, N. Y., Ballston Artesian Lithia Well, N. Y.,] are distinguished through carbonic acid.

The sodium chloride waters, in contrast to the sulphate waters, do not seem to have an influence upon the large abdominal glands (liver, pancreas), but only upon the stomach and intestines.

The essential action of the sodium chloride salts is, in my opinion, due to a stimulation of the glandular secretion, improvement of appetite, and removal of gastric mucus.

Specifically typical is this favourable influence in cases of *incipient gastritis associated with HCl deficiency,* in which disturbances are manifested subjectively through pressure and fulness after eating, salivation, nausea, tendency to vomiting, or actual vomiting, and objectively through a more or less reduced HCl production and formation of mucus. In these cases, provided a secondary process is not present, the use of sodium chloride waters (perhaps combined with hydrochloric acid) is of excellent effect. As I frequently observed, a decided increase of HCl production, with a subsequent better chymifaction and disappearance of the thick and ropy mucous masses, is the result. This experience, made first by v. Sohlern[19] and myself, has recently been confirmed by Dapper; to be sure, Dapper[20] also obtained in a small number of cases with superacidity a decrease of the disturbances as well as a diminution of the acidity.

Although I could occasionally observe favourable subjective results in cases of gastritis of long years' duration, and with constant loss of HCl and simultaneous defect of ferments, I could not observe any influence upon the chemical functions.

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[18] L. Wolff, Zeitschrift für klinische Medicin, 1889, Bd. 16, S. 256.

[19] v. Sohlern, Berliner klinische Wochenschrift, 1897, No. 21.

[20] Dapper, Zeitschrift für klinische Medicin, 1896, Bd. 30, Heft 3 and 4.

In the majority of these cases, sodium chloride waters (in medium and large doses) have also a favourable effect upon gastro-enteric peristalsis, and, at least during the time the waters are taken, the bowel movements become regular and flatulency is diminished. If large quantities of sodium chloride waters are introduced, thin fluid or watery evacuations may occur, which may cause a loss of fat and proteids. In small doses (1 to 1½ cups a day) they constipate.

The conclusion, therefore, is that cases of *increased chemical functions* are not adapted for sodium chloride waters. I was enabled to observe this fact in patients who, after an inefficient "cure" in Kissingen, came to consult me. The same observations were also made by v. Sohlern,[21] in disagreement with Dapper, and my views have also not changed in spite of the above-mentioned observations of Dapper. The same evidently refer to conditions of nervous, irritative dyspepsia, in which the administration of Rakoczy water is of secondary nature when compared to a rational diet. Whether the acidity increases or decreases, does not matter in such cases, in my opinion; important only is the improvement of the nutrition and of the bodily strength. In cases of pyrosis hydrochlorica, ulcerative superacidity, and gastritis acida, I am convinced that drinking of Kissingen waters is not to be recommended.

I am equally convinced on a further point: *atonies and dilatations of the stomach are no objects for "drinking cures" of sodium chloride water.* In the former there exists, as a rule, at least in comparatively recent cases, hyperchlorhydria. If the patient is allowed to drink small doses an undesired effect is obtained: the acid secretion continues to increase. If large doses are chosen, a further superextension of the stomach occurs. I could prove this argument with a great number of clinical histories. [It is in these very same atonic conditions of the stomach, that frequently the greatest mistakes are made by physicians sending their patients to watering places. In the greatest majority of the cases an examination of the stomach-contents and of the gastric motor function has not been made, and nevertheless the patients are advised by their physician to drink three to four glasses (750 to 1000 cubic centimetres) of the mineral water three times a day, so that the patient drinks from two to three litres of water, whereby the possible existing atony becomes almost dilatation. I had occasion to observe repeatedly cases of this kind at Bedford Springs, Pa.]

Still less adapted are sodium chloride waters, or, for that matter, any mineral water, to dilatation due to organic stenoses or myasthenia. In these cases our chief object is to provide favourable conditions for the exit of the fluids; every superfluous introduction of water, whether it contain sodium chloride or sodium sulphate, must accordingly be avoided.

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[21] v. Sohlern, *l.c.*

Regarding gastric ulcer, Carlsbad has acquired such a dominant reputation that it would be labour lost to try to make changes in it. Yet I like to believe that mild sodium chloride waters might be equally indicated for some cases of chronic gastric ulcers, particularly for those not associated with superacidity; it is true, I have no experience of my own in this matter. The use of sodium chloride waters in cases of carcinoma, even in their early stages, is, of course, to be avoided.

As to gastric neuroses, sodium chloride waters are not more adapted to their treatment than sodium sulphate waters are. To be sure, this is correct only in those cases in which the various gastric functions have been proved to be normal. In mixed cases, in which nervous symptoms are only more prominent in the general picture of digestive troubles, and if the latter do not form a contra-indication, sodium chloride waters may be advantageously employed.

The most important sodium chloride springs and their composition are to be seen from the following table:—*

In 1000 Parts of Water.	Table Salt	Carbonic Acid
Nauheim (Kurbrunnen)	15.42	995
Neuhaus (Bonifaciusquelle)	14.77	1138
[Ballston Artesian Lithia Well, New York	12.49	1647]
Also-Sebes	11.77	
Salzschlirf (Tempelbrunnen)	11.40	1029
Salzschlirf (Bonifaciusbrunnen)	10.20	873
Homburg (Elizabethbrunnen)	9.86	1039
Schmalkalden	9.34	115
[Saratoga, N. Y., (Hathorn)	8.60	1023]
Münster a. Stein (Hauptbrunnen), Temp. 31° C.	7.90	
Pyrmont (Salzquelle)	7.05	954
Wiesbaden (Kochbrunnen), Temp. 68° C.	6.83	200
Mergentheim	6.67	297
[Saratoga, N. Y., (Congress)	6.65	1270]
Bourbonne les Bains, Temp. 50° C.	5.98	228
Kissingen (Rakoczy)	5.82	1305
[Saratoga, N. Y., (Kissingen)	5.64	1421]
Soden (Milchbrunnen)	5.42	951

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* We have not listed the waters containing less than two per mille table-salt among the sodium chloride waters, as they hardly deserve this designation.

Bourbonne les Bains, Wiesbaden, and Münster a. Stein, among the mentioned sodium chloride waters, represent *sodium chloride thermal waters.*

5. *Chalybeate Waters.*—These are frequently employed in the treatment of chronic dyspepsia, in the form of so-called alkaline acidulous waters, for the genuine chalybeate waters are badly borne in cases of pronounced dyspepsia. First deserving mention are Franzensbad and Elster, because either one has, besides the chalybeate waters, an almost pure sodium sulphate spring, the use of which is of great value on account of habitual constipation concomitant with almost all anæmic conditions. To this class also belong the iron waters of Reinerz and Cudova in Silesia, Rippoldsau, [Petersthal, Freyersbach] in Baden, Schwalbach, Bartfield, and many others.

I occasionally observed excellent results in anæmia with cardialgic and other dyspeptic conditions, from the use of the Eger-Franzensquelle and the iron springs of Elster. [In America, this class of waters is excellently represented by various springs, such as Iron Spring, Paducah, Ky.; Dawson Springs, Ky.; Estill Springs, Ky.; Chalybeate, Bedford, Pa.; Church Hill Alum Springs and Rock Ennon Springs, Virginia; Vichy Springs, New Almaden, Cal.; Washington Springs, Saratoga, N. Y., etc.]

6. *Lime waters,* among them, above all, those containing calcium carbonate, are used in those cases in which, besides dyspeptic conditions, a disposition to *diarrhœa exists,* particularly in catarrh of the small intestines. Quite particularly adapted are these waters also for table waters, with addition of wine, brandy, etc. The most important springs of this kind are Wildungen (Königsquelle), Driburg (Hersterquelle), Lippsprings (Arminius), Rappoldsweiler (Carola), Coburg (Marianna), Marienbad (Rudolf), and Krynica[22] (Hauptquelle). [In America, we may mention among other springs, Saratoga Vichy Spring; Ephrata, Pa.; St. Louis Spring, Michigan; Bethesda Springs, Wisconsin.] The said springs are cold waters, but in cases of diarrhœa should be heated before drinking.

7. *Bitter Waters.*—These are to be considered for the treatment of gastric affections only in so far as the latter represent the consequences of habitual constipation or abdominal plethora, hæmorrhoidal affections, etc. In primary disturbances of digestion, their use is to be restricted as much as possible. Directly contra-indicated is the use of bitter waters in cases of gastric and duodenal ulcers. I observed a fatal effect in a case of gastric ulcer in the practice of a colleague after use of bitter water; in other cases decided deterioration of the condition occurred. Also in all gastro-enteric affections which lead or may lead to adhesions, as, for instance, in typhlitis

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[22] Piatkowski, Wiener Klinische Wochenschrift, 1898, No. 1.

stercoralis [appendicitis], bitter waters ought to be supplanted by mild purgatives (castor oil, rhubarb, cascara) or, still better, by dietetic laxatives. [To this class of bitter waters belong, to mention a few, Pullna, Bohemia; Friedrichshall, Germany; Hunyadi Janocz, Hungary; Epsom Spring, England; Crab Orchard and Estill Springs, Kentucky; Bedford Mineral Springs, Pa., etc.]

In the majority of cases of disturbances in the alimentary tract, systematic bathing is combined with systematic drinking. *Salt* and *mud* (Moor) baths are particularly well liked in these cases, but in some cases the simple akratothermæ and iron baths may exhibit favourable effects. The former are especially appropriate in cases of exudates in the gastro-enteric area, peritonitic adhesions, etc. Kisch[23] highly recommends *moor cataplasmata* (*mud poultices*) in gastric neuroses with increased sensitiveness. I recommend hot mud poultices, particularly in gastric ulcer and cholelithiases. The mud baths of Marienbad, Franzensbad, and Elster are the best known and the most successful [Mineral Spring Mud Baths, Attica, Indiana, are frequently used in the middle West and are good.] Salt baths are usually employed there, where the systematic drinking of the saline waters can be combined with them; resorts with salt bath are not infrequent and are often of great benefit.

The above discussions lead to the conclusion that three groups of gastroenteric affections are in general *not* adapted for the use of mineral waters: 1. Conditions of advanced muscular relaxation of the gastro-enteric canal, as well as of gastric dilatations due to stenosis or myasthenia. 2. Neuroses of the alimentary tract. It shall, however, not be said that in special cases neuroses may not do well in watering places which owe their reputation to the mineral water springs; but I should prefer to ascribe this result more to the favourable external circumstances, the change of climate, the suitable diet, the employment of other curative measures, which nowadays are at the visitor's disposal in every health resort.* I speak here only of those neuroses in which objective examination does not exhibit any hint of the presence of functional or anatomic changes. 3. *Systematic drinking of mineral waters is to be omitted on principle, in cases of carcinoma,* as even a palliative effect is not to be expected. Carcinoma of the stomach is preferably reserved for domestic attention or for the hospital. The same is to be said also of affections suspected of cancer.

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* [Unfortunately such is not the case in a large number of American watering places. Physiologic-therapeutic, and particularly dietetic methods are very much neglected in health resorts in this country; it is partly the fault of the physicians, partly the fault of the proprietors and managers of the hotels, and especially the fault of the indifference of the communities, which prefer races, sports, etc., to hygienic-therapeutic improvements. There are in this country quite a number of superb places which could easily rival or even excel European resorts, if the right spirit were present.]

[23] Kisch, Balneo-, Hydro- und Klimatotherapy, Wien und Leipzig, 1883.

Where mineral water cures are not indicated, there remain two great and excellent curative factors, namely: the climatic health resorts (mountain, forest, etc.) and the sea-shore and ocean baths. It is sometimes very difficult to make a selection between these two. Special indications can hardly be established, and only a few general points may be emphasised. Change of climate, especially mountain air, is adapted in all cases in which the neurosis is evidently due to unsuitable sedentary life, and furthermore to mental and bodily over-exertions. We often hear from the patients themselves where they belong: a careful examination shows that their troubles are immediately removed as soon as they have left the dust of the cities and live in quiet surroundings.

To the sea-shore and ocean baths, on the other hand, such individuals should go as manifest pronounced nervous disturbances in the alimentary tract. Among these are loss of appetite or perverse appetite, depression, languor, lassitude, constipation, loss of weight. According to Lindemann,[24] cases of gastric and enteric atony and enteroptosis are also indicated, but not patients with severe irritative conditions of the stomach. Likewise contra-indicated is the sojourn at the sea-shore in the majority of organic gastric diseases, particularly those associated with gastric irritation, such as gastrectasis, gastric ulcer, chronic gastritis, etc.

But even in the suitable cases, we must consider whether there are not, besides the nervous troubles, some objectively demonstrable functional changes which require a strict diet. As the majority of the sea-shore resorts, particularly those situated by the North Sea [likewise those on the Atlantic coast of America] do very little or nothing at all in regard to a rational diet, it demands a careful and critical selection.

An indispensable curative factor in some cases is the treatment in appropriate *sanatoria*. The significance of the treatment in sanatoria rests upon the possibility of watching the patient continuously, to regulate his diet in pursuance of progressing examinations of the gastro-enteric function, and to give him corresponding directions for his subsequent conduct. Treatment in sanatoria is adapted to such patients as require a suitable diet, or in whom a suitable feeding cannot be carried out for *external* reasons. Also where the application of physical curative methods (massage, electricity, hydriatic treatment) is indicated, the treatment in an institution is preferable to the ambulant treatment. In individual cases we consider the sanatorium treatment especially indicated in recalcitrant, frequently-recurring gastric or duodenal ulcers; also in ectasia of the stomach, in certain neuroses (nervous vomiting, gastralgia, nervous dyspepsia), in enteroptosis, in enteric catarrhs at advanced stages, finally, in persons who have not enough energy and will-power to submit to a strict dietetic treatment. Moreover, scientifically well-conducted sanatoria are very well adapted for the making of diagnosis in obscure and complicated cases.*

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[24] Lindemann, Deutsche Medicinal-Zeitung, 1897, No. 26, 27, 28.

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* By appropriate sanatoria I do not mean large sanatoria which serve for all possible medical branches, and in which the surgeon and gynæcologist, the dermatologist and neurologist, and in addition, also, the gastro-enterologist are concurrent, but sanatoria of smaller extent, which serve exclusively to the rational feeding of sufferers from gastric diseases of the stomach and intestines, and consequently make use of a kitchen suitably conducted, solely for patients of this kind. There are many sanatoria which do not conform to these requirements. It is also a matter of course that the personality of the director of the institution, his scientific ability and practical experience are to be considered in the selection of a sanatorium. [If this brief discussion on sanatoria applies to those in Europe, it certainly is applicable also in America, where there is almost an absolute want of institutions of this kind in any city. Experiments in this direction have been tried, but very frequently failed on account of either lack of interest, or perhaps, also, of narrow-mindedness of some members of the medical profession; I do not think it is the fault of the patients, who would be only too willing to follow the advice of their physicians. In many cases it is the wrong attitude of the directors and managers of such institutions, who will not allow the attending physicians a participation in the medical treatment of their patients.]

CHAPTER XI.

Physical Curative Methods.

Massage of the Stomach.

The fact that motor weakness and conditions of relaxation in the digestive tract are *very frequent* affections, attributes a certain aiding part to the mechanical treatment.

The train of thoughts for this treatment is as follows:—

1. The inert and paretic musculature is strengthened, or ligamentous adhesions are loosened or relaxed.

2. The contents which remain for an abnormally long time in the alimentary tract are propelled.

While in the course of years massage of the intestines has been generally adopted and has proved itself as practical, such is not the case with massage of the stomach. This depends on the fact that accurate indications are missing, so that under some circumstances the mechanical treatment may cause direct injury.

In general, massage is indicated:—

1. In those gastric affections in which there is a simple atony of the muscularis or a relaxation of the ligaments and a consequent deficient support of the organ (gastroptosis).

2. In certain forms of gastrectasis due to pyloric stenosis. Favourable results in such cases were reported by Zabludowski[1] some time ago. The cases adapted for this treatment require a careful election in so far as in all pyloric stenoses associated with marked *fermentative processes* massage may be injurious because the fermenting masses are propelled into the intestines and thus offer far more favourable conditions for growth of bacteria.

3. In certain forms of nervous dyspepsia, particularly the sensory forms (sensations of pressure, pain, irritable stomach, etc.). Here also not every case is adapted for the mechanical treatment; a tentative trial has perhaps to decide.

4. In all cases in which there exists a primary intestinal atony which leads to secondary disturbances of the gastric functions. The usefulness of mechano-therapy is proved in numerous cases of this kind.

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[1] Zabludowski, Berliner klin. Wochenschrift, 1886, No. 26 *et seq.*

5. Favourable chances are offered through the application of massage in all localised exudative processes, adhesions, inflammatory thickenings, etc.; in such cases, however, great caution and close examination of the cases in question are indispensable.

Massage is *contra-indicated* in all *recent* cases of ulcer with adhesive connection with neighbouring organs, in which cases even its cautious application may cause a perforation of the ulcer into a neighbouring organ, with its well-known disastrous effects. If stomach or intestines are filled with ingesta or with air, great caution is necessary. "If the gastric muscles are spontaneously very active, if the peristaltic movements are pronounced and frequent, or sometimes as if in a tetanic condition, and if the stomach felt by the hand feels somewhat as a contracted uterus after birth, hands off!" (Zabludowski.)

It would be superfluous to mention that massage is to be avoided in all cases of tumour formations, whatever their location or their type may be, if I had not repeatedly had occasion to observe the sad effects of such a polypragmasy, unfortunately sometimes on the part of physicians, but particularly on the part of so-called "nature-physicians" [osteopaths, etc.]. *Apropos,* I wish to mention that massage is also to be avoided in cholelithiasis, for in various cases I observed severe attacks immediately after abdominal masage.[2]

As regards the technique of the massage, we are compelled to confine ourselves here only to a few general remarks. It is evident that in the different affections the mechano-therapy has various objects. In many cases (pyloric stenosis) the gastric contents are to be pushed partially or totally through the pylorus by means of mechanical measures, and the passage is thereby to be forced similarly as it is done in an acute way by the surgical digital divulsion. In other cases the mechano-therapy is to invigourate the muscles of the gastro-enteric canal and to enable it to gain an increased dynamic function. In such cases it is, of course, best to massage the stomach when it is empty, that is, in a relaxed condition. The same is to be said also in the above-mentioned cases of nervous dyspepsia. The direction, repeatedly and urgently given by Cséri,[3] that the stomach ought to be always massaged when full, may have its advantages in some cases, but certainly must not be generalised.

It is difficult to establish the effect of massage with any degree of certainty, because usually it is employed in combination with other remedial measures, particularly with dietetic methods. Indeed, the question whether the muscles are stimulated *permanently* or temporarily, or to any degree at all, is not answered with certainty. According to Penzoldt's[4] investigations on healthy physicians, it appears "that the effect of massage upon the normal digestion depends upon the individuality of the person or upon the incidental method of massage. At least it may be imagined that in one person the resistance of the pyloric muscles may be easier overcome than in another; likewise, that under favourable conditions of the abdominal walls and of the fulness of the stomach (etc.), we may succeed through a lucky grip in expressing the stomach,* while a regular result is not to be obtained."

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[2] Boas, Münchener medizinische Wochenschrift, 1902, No. 17.

[3] Cséri, Wiener medic. Wochenschrift, 1894, No. 46-48.

There exist also investigations on the influence of gastric massage upon the secretion, according to which the same is said to influence favourably the hydrochloric acid secretion (Reed,[5] Cautru[6]). Accordingly, massage is not indicated in cases of superacidity.

Only a slight value is to be ascribed to these investigations, as we have to deal only with the effect of massage done but once or repeated at short intervals, but not with a final result, or a systematic use of massage—which latter is the chief point. According to my conviction, gastric massage, which, moreover, is usually combined with abdominal massage, acts predominantly upon the intestines, and in so far as the intestinal peristalsis is favourably influenced, the gastric motility is also indirectly favourably influenced.

In localised inflammations or adhesions, it is evident that the corresponding portion of the stomach alone or principally will be the subject of the treatment.

According to the object to be accomplished through the mechanical treatment, the technique must of course vary. In the passive evacuation of the stomach through the pylorus, Zabludowski recommends inserting the right hand deeply and grasping on the left side a portion of the fundus between the extended thumb and the four other fingers. Thus we get some kind of a fold which contains abdominal walls and gastric wall with the chyme contained in it. By pushing or slinging movements produced at the fold the gastric contents are, so to say, thrown towards the pylorus. [Hemmeter, in his book on "Diseases of the Stomach," reproduces three illustrations from Penzoldt and Stintzing, "Handbuch der Therapie," and gives the following instructions for improving the muscular tone of the empty stomach: The masseur places himself to the right of the patient, who must lie on his back, with knees slightly flexed.

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* More correct, "contents of the stomach."

[4] Penzoldt, Festschrift zur Feier des 80. Geburtstages Sr. Königl. Hoheit des Prinzregenten Luitpold von Bayern, 1901, P. 10.

[5] Reed, International Magazine, January, 1898.

[6] Cautru, referred to in Virchow-Hirsch, Jahresbericht, 1900, Bd. 2, P. 247.

First movement (*a*): Insert the left hand slowly and gradually deeply under the left arch of the false ribs, under and past the edge. To increase the pressure, gently press the right hand firmly on the left. Second movement (*b*): Now describe small circles with the hands thus arranged, progressing slowly from the pylorus to the fundus. Third movement (*c*): Perform strong vibratory movements toward the depth with the finger-tips, while *a* and *b* are being executed. Fourth movement (*d*): Knead the stomach between thumb and four fingers, and in conclusion execute stroking passes, with extended four fingers, from left to right.]

In more progressive cases, in paresis of the muscles, the stomach is pressed against the spinal column and is thus divided into two parts, one belonging to the fundus region, the other to the pyloric portion. "The column of chyme, enclosed in the pyloric portion and to be considered as an incompressible body, serves, when pressed from outside, as a bougie for a dilatation of the stenotic passage" (Zabludowski). In the real kneading (pétrissage, and massage à friction), Zabludowski advises to insert the fingers as deeply as possible into the portions to be worked upon. "Knead the stomach in a horizontal direction, as you would knead dough, with expansive equal motions of both hands, and alternate the latter manipulation with circular motions by the one hand and longitudinal motions with the other hand."

Occasionally Credé's method is applied, but with the difference, that in regard to the expressions of the placenta the pressure is exerted longitudinally, and as to the stomach, transversally.

Less adapted, according to Zabludowski, is the true stroking proper, effleurage and tapotment. But, relying on my own experience, I did not reach the same negative view as the said author did. To abandon tapotment entirely for fear of a reflex effect upon the heart (Goltz's experiment), there is, as far as I can see, no reason for it.

The duration of one treatment should be ten minutes, that of the single manipulation should not be more than two or three minutes.

Massage of the stomach may suitably be combined with massage of the other part of the abdomen, particularly of the intestines, if an indication for it exists.

Frequently apparatus in forms of globes and rolls have been recommended for self-massage and are well known to the general public. Possibly results from this method might be ascribed less to the method than to auto-suggestion.

Massage may, as Wegele[7] recently recommended, be combined with application of medicines. The fluid is drank or introduced by the tube. Patient is in recumbent position, and the stomach is stroked and rubbed in order to bring the medicine in closest contact with the mucous membrane. Wegele employs physiologic salt solutions, 1 per cent. ichthyol solution, 1½ per cent. Carlsbad salt solution, 5 to 6 per cent. bismuth-suspensions, 1 or 2 per mille silver nitrate solution with subsequent salt water solutions, decoction of bitter remedies (amara) and disinfecting solutions. As adapted for this procedure Wegele considers the various affections of chronic hyperæmia (?) and irritation of the mucous membrane without regard to increase or decrease of the chemical functions. He also recommends the method, which, however, has as yet not been substantiated, in hyperæsthesia of the gastric muscosa, and in cases of purely nervous gastralgia.

In conclusion, I may remark that gastric and abdominal massage is best applied by the physician himself, and ought not to be performed by laymen.* There are difficulties, however, against putting these requirements in practice, in so far as massage has been taught only in the last few years at our universities, and that at the present time only a few physicians are so familiar with the technique that they could master the rational and approved methods.

The Electric Treatment in Diseases of the Stomach.

It is true, we are still lacking in knowledge regarding the manner of action of the electric current, and the experimental investigations hitherto made are full of contradictions. Till these differences are cleared up, we must adhere to the clinical experience, which, in general, speaks in favour of electric treatment.

The use of electric treatment in gastric diseases refers, as otherwise in electro-therapy, to the faradic as well as galvanic current, and it is said that the former acts more upon the motor function, and the latter more upon the secretory apparatus; some authors also suppose an influence upon the absorption.

The physiologic foundations of these doctrines, as far as we can review them at the present time, are based on following observations and experiments:—

(*a*) *Regarding the Motility.*—After earlier authors (v. Ziemssen, Rossbach, Lüderitz) had already established that stimulation of the gastric fundus by means of faradic currents produces contractions to a rather slight extent, Meltzer[8] substantiated and advanced these statements through thorough investigations on dogs, finding that however strong the faradic currents acting upon the fundus portion of the stomach may have been, contractions never occurred, and it was only when the pyloric section was approached that Meltzer succeeded in obtaining contractions, the strongest being at the pylorus itself. This effect could not be gotten from the corresponding area of the mucous membrane. When, after exposure of the stomach, an electrode is put upon the abdominal wall near the stomach while the other is put on the back or introduced into the stomach, even with the strongest currents only a contraction of the abdominal muscles occurred, but never one of the stomach. These experiments are contradictory to Einhorn's experiments on frogs, which are supposed to demonstrate contractibility of the fundus region; but these experiments, too, exhibited only a very slight activity of the fundus region on stimulation through the faradic current. Quite analogous results were shown through the more recent investigations of Goldschmidt[9] and Goldbaum,[10] who could not demonstrate either a secretory or a motor influence upon the stomach through faradic or galvanic currents. Herewith an opinion by the past master of clinical medicine, v. Ziemssen,[11] is to be considered as proved; he expressed himself as follows: "I cannot designate as principally correct the view that, regarding the electrisation of the stomach, we have to deal chiefly with the tendency to effect contractions of the gastric muscularis and diminution of an ectatic stomach."

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* Hoffa, in his excellent book, "Technik der Massage," says: "I claim that every lay-masseur who performs massage independently, is and remains a quack."

[7] Wegele, Zeitschrift für diätetische und physikalische Therapie, 1899, Bd. 2, P. 257.

[8] Meltzer, New York Medical Journal, June 15, 1895; Archiv für Verdauungskrankheiten, 1897, Bd. 3, Heft 2.

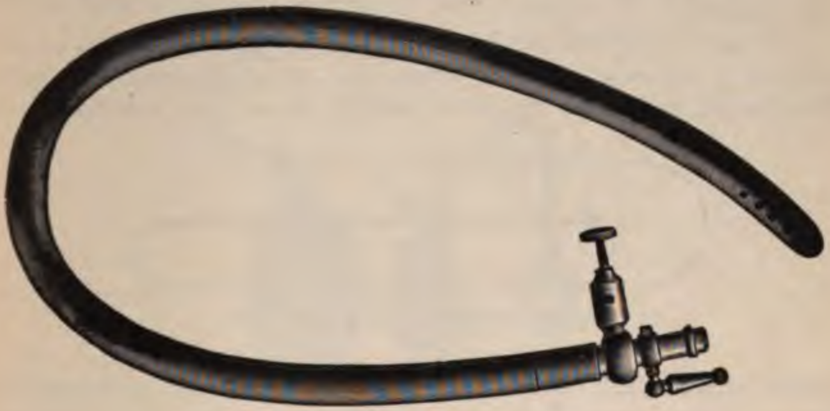

Fig. 46.—Electric Stomach-tube. (Boas.)

(*b*) *Regarding the Secretion.*—As regards secretion also, the results of investigation are difficult to explain. v. Ziemssen and Rossi had effected, in experiments on animals, increased secretion through either kind of current. The same result was obtained by Hoffmann in experiments on human beings who were treated with galvanic currents, and that intraventricularly. In contrast to this, neither Goldschmidt nor Goldbaum was enabled to prove any recognisable influence on application of the galvanic or faradic current in the above-mentioned experiments made likewise on human beings. The question, therefore, has not yet been decided.

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[9] Goldschmidt, Deutsches Archiv für Kline Medicin, 1896, Bd. 56.

[10] Goldbaum, Archiv für Verdauungskrankheiten, 1897, Bd. 3, Heft 1.

[11] H. v. Ziemssen, Die Electrizität in der Medicin, 1887, P. 445.

Moreover, according to my opinion, not much importance must be ascribed to the fact of secretory or motor increase during the electric stimulation. For practice does not demand a stimulus which only occurs during the phase of the electric treatment and quickly disappears, but a permanent favourable influence. In this regard no experiments are existing, and in the meantime it will be advantageous not to expect too much as to the permanent effect of the electric current.

(*c*) *Regarding the resorption*, only a few experiments of Einhorn have been recorded, which were made on the basis of Penzoldt's potassium iodide test, and showed an improvement of resorption on intraventricular faradisation.

(*d*) *Regarding the sensibility*, v. Leube, and more recently Einhorn, observed favourable results from galvanisation, and that especially in obstinate gastralgias.

Clinical experiences show more accord as to the local electric treatment. Before we discuss the same, a few remarks on the technique may precede them.

We may employ the galvanic and faradic current, either of them percutaneously or, more correctly expressed, extra-abdominally and intra-abdominally. As regards the latter method, it was first introduced into practice by Kussmaul, and was later extended by Bardet also to the galvanic current. The method in its simplest form consists of the fact that a metallic wire which is connected with the induction or constant apparatus, is put into a rubber tube and swallowed. From this have developed in recent years quite a number of "electric stomach-tubes," which, however, are all based upon the above-mentioned principles and exhibit only non-essential technical advantages. The electric stomach-tube, illustrated in Fig. 46, and advanced by myself, and which is distinguished from others by the fact that it contains a platinum spiral and synchronously allows affluence and effluence of waters, has proved itself very excellently adapted for the endofaradic and galvanic treatment for several years past. Much to the purpose appears to me also the gastric electrode (Fig. 47) constructed by Wegele.[12]

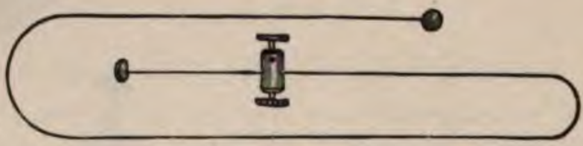

Fig. 47.—Wegele's Gastric Electrode.

There are, however, still other numerous devices which may, of course, likewise be of service. Before the application of the electric treatment, about 300 to 500 cubic centimetres of water or physiologic salt solution are introduced into the stomach.

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[12] Wegele, Therapeutische Monatshefte, 1895.

As a whole, the experiences about the intraventricular electrisation vary very much. Favourable results were observed by Kussmaul,[13] unsatisfactory results, especially in dilatations, by v. Ziemssen.[14] Erb[15] reserves the same only for rare cases and prefers, according to the procedure of de Watteville, the combined galvano-faradisation. Ewald (*l.c.*) favours the intra-ventricular treatment and declares that the unfavourable observations of v. Ziemssen were made at a time when the technique of the gastric treatment had not reached the acme of modern times. My experiences likewise do not speak in favour of the endogastric application, although I do not go so far as to replace the same, under all circumstances and in all cases, by the percutaneous method. Thus, for instance, it seems to me very appropriate in cases of cardiaparesis due to central nervous disturbances or to neurasthenia, in which the mentioned paresis represents, according to my observations, a frequent and troublesome complication.

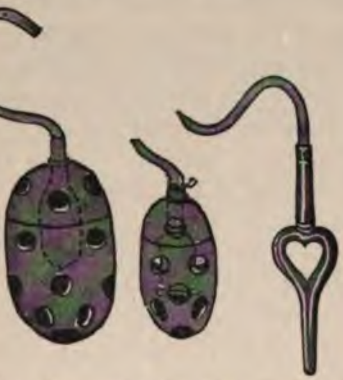

Fig. 48.—Einhorn's Apparatus for Endogastric Electrisation.

Another method of endogastric electrisation has been described and recommended for the practice by M. Einhorn.[16] This author constructed an electrode according to the principle of the already-mentioned "gastric bucket," which is swallowed by the patient and thus by itself enters the stomach (Fig. 48). The silk thread of the bucket is replaced by a very narrow rubber tube, through which very fine wires lead to the battery; the end-piece of the electrode consists of a hard-rubber capsule with multiple perforations, in which a metal button connects with the tube.

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[13] Kussmaul, Archiv für Psychiatrie und Nervenkrankheiten, 1877, Bd. 8, S. 205.

[14] v. Ziemssen, Ueber die physikalische Behandlung chronischer Magen und Darmkrankheiten, Leipzig, 1888, S. 10.

[15] Erb, Handbuch der Elektrotherapie, 1882, S. 622.

[16] M. Einhorn, Berliner klinische Wochenschrift, 1891, No. 23.

The patient, preferably on an empty stomach, drinks one or two glasses of water, and swallows likewise, with the aid of water, the hard-rubber capsule, whereby the electrode enters the stomach. The other electrode is applied either to the back at the left of the seventh thoracic vertebra, or to the epigastrium, or it is held in the hand. Einhorn considers this method, about which I have no experience, as "extremely simple and convenient for patient and physician," and as easy to be performed as the percutaneous electrisation. In contradiction, Ewald[17] pointed out that the introduction of Einhorn's deglutable electrode frequently meets with difficulties, and employs, accepting the form of the said electrode, instead of the conducting wire, a common stomach-tube (Charrière 13), which is suitably connected with the electrode.

As regards the technique of the extra-abdominal electrisation of the stomach, it is to be mentioned that, according to v. Ziemssen's[18] proceeding, the large plate electrodes are decidedly preferable to the usual small ones. v. Ziemssen places the larger one (600 square centimetres) upon the anterior abdominal wall, in the direction from the pylorus to fundus, and the smaller one (500 square centimetres) from the fundus to the vertebral column, corresponding to the position of the stomach. "The distance between the edges of the electrodes situated over the fundus of the stomach should not amount to more than one or two centimetres. The strength of the current, corresponding to the area of the electrode, must be rather considerable. At the induction current as well as at commutations of the constant current (strength of current, 10 to 20 milliampères), marked contractions of the abdominal muscles, etc., must occur, which, through contractions of the abdominal and dorsal muscles, produce motions of the trunk and perhaps also contractions of the diaphragm, without being markedly painful" (v. Ziemssen). The single treatment should not last more than ten minutes. Massage may be suitably combined with electricity. Very well adapted to this is the *"electric roll,"* in the employment of which, for instance in atonic conditions of the gastro-enteric canal, I observed some astonishing results.

Likewise of good service in atony of the gastro-enteric canal was the treatment by means of the *"electric brush,"* particularly as regards the improvement of peristalsis.

As regards special indications for the application of electricity, first of all the frequent defects of the mechanical gastric function are to be considered, as well as relaxations of the muscles and their sequelæ; in such cases Einhorn and the majority of others recommend faradisation, but Rosenheim *recommends galvanisation.* No good results are obtained in pyloric stenosis, because the causal indication, namely, the removal of the obstacle, is of course not affected. Perhaps the gastric electrisation plays an important part in the after-treatment of (cicatricial) stenoses removed through surgical operation. The faradisation of the abdomen sometimes effects great results in habitual constipation. Evidently these results refer only to cases due to muscular sluggishness, while an essential effect is not to be expected in the presence of mechanical obstacles. Some physicians replace the abdominal electrisation with the introduction of an electrode into the rectum, in some cases with great or even astonishing results. I myself, after having formerly had unfavourable results with bad electric tubes, consider this method as very effective, especially in atony of the lower sections of the colon.[19]

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[17] Ewald, Berliner klinische Wochenschrift, 1892, No. 26-27.

[18] v. Ziemssen, *l.c.*

The *second* indication is given in the great number of gastric neuroses. It is difficult to tell which cases offer individually favourable chances for the electric regimen. Frequently a satisfactory result is due to the combined application of various curative measures.

v. Leube[20] observed excellent results from the application of the constant current in gastralgia, Semmola[21] likewise in nervous vomiting of hysteric and pregnant patients. Rosenthal[22] employs general faradisation in asthenic dyspepsia, beside galvanisation of the sympathetic nerve and of the vertebral column; Burkart[23] reports favourably about the faradic current in gastralgia and nervous and neurasthenic dyspepsia; Rosenheim[24] and Brock[25] also praise galvanisation in gastric neuroses, especially in sensory irritative symptoms. According to Goldschmidt's most recent investigations, there is no essential difference between endogalvanisation and endofaradisation; it seems, however, that galvanisation (anode in the stomach) is more adapted for painful gastric disturbances, and the faradisation rather for the functional troubles. Finally Caron,[26] and more recently Goldbaum, observed very remarkable results from intraventricular faradisation in cases of stubborn habitual (nervous) vomiting. In recent years it was Einhorn[27] particularly who studied closely the technique and indications of the intraventricular faradisation and galvanisation. The same established the following indications:—

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[19] Boas, Diagnostik und Therapie der Darmkrankheiten, 1901, 2 Aufl., S. 178.

[20] v. Leube, Ziemssen's Handbuch, Bd. 7, Theil 2, S. 205.

[21] Semmola, Gaz. med. ital. Lombard, 6/78.

[22] Rosenthal, Magenneurosen und Magenkatarrh, sowie deren Behandlung, Wien, 1886.

[23] Burkart, Zur Pathologie der Neurasth. gastrica, Bonn, 1882.

[24] Rosenheim, Berliner Klinik, 1895, Heft 71.

[25] Brock, Therapeutische Monatshefte, June, 1895.

[26] Caron, Thèse de Paris, 1891.

The direct gastro-faradisation exhibits manifold usefulness in the majority of gastric effections (cancer excepted); very distinct is the effect in gastric atony, whether the amount of acid is too high or too low. Further, Einhorn recommends endofaradisation in pyloric or cardiac relaxation. Direct galvanisation was employed with great effect in cases of stubborn gastralgia, whether due to nervousness or to ulcer cicatrices. Moreover, Einhorn observed a favourable effect in heart affections which are associated with gastralgia.

Einhorn gives the following directions for the intraventricular faradisation and galvanisation:—

1. *Faradisation.*—Single treatment to last ten minutes: First, large plate electrode to the epigastrium for five minutes; after that, small sponge electrode, transversally over the gastric region; in constipation, simultaneously region of the colon (starting in the ileo-cœcal region, then ascendent, transverse, descendent colon); then to the left, around the fundus region towards the back, seventh vertebra (one minute), then again to the front, stroking the gastric region upward and downward (two minutes), gradual ceasing of the current. The current must be so strong that pronounced contractions are produced, but not so strong that pain is provoked.

2. *Galvanisation.*—Negative pole in the stomach, small sponge electrode. Duration, eight minutes. First, two minutes at the spot below the ensiform process (slowly increasing); three minutes' stroking upwards and dôwnwards in the gastric region, then going towards the back (one minute at the seventh vertebra); return to the front (one minute), the minute below the ensiform process, decrease the current slowly, and cease. Strength of current, 15 to 20 milliampères.

The Hydriatic and Thermic Treatment of Diseases of the Stomach.

Hydrotherapeutics forms an essential part of our treatment of digestive disturbances. The simplest form is the well-known Priessnitz Umschlag (wet pack), the usefulness of which in many affections of the gastro-enteric tract is beyond any doubt. As every practitioner knows from experience, the application of this very simple pack requires a particular technique which makes it necessary to give exact directions to the patient. If the wet towel is not satisfactorily covered, cold from evaporation develops, whereby the body and the internal organs are constantly exposed to temperature changes which are felt as exceedingly troublesome, particularly by sensitive patients.

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[27] M. Einhorn, New Yorker medicinische Wochenschrift, 1891, No. 9; Deutsche medicinische Wochenschrift, 1893, No. 33-35; Zeitschrift für klinische Medicin, 1893, Bd. 23, Heft 3 and 4; ferner: Diseases of the Stomach, New York, 1897, S. 136, etc.

Accordingly, absolute covering through wax or silk taffeta and a woolen cloth is indispensable.

The *methodically* carried-out hydrotherapeutics has at its disposal the most different variations, which for the most part originate in empiricism. To them belong the rubbing, slapping with moist towels, wet packs, cold, warm, tepid and chilled full and half baths (25° to 20° C.), douches, etc. Rosenthal recommends the Scottish douche (with water of 28° C., followed quickly by water of 12° C.) in dyspeptics with troublesome pneumotosis and eructations of air; I also observed quite satisfactory results in some cases of this kind.

Important aids, likewise to be mentioned here, are the hot poultices, which, first introduced into the treatment of gastric ulcers by v. Leube, have since been generally adopted by the medical profession. Linseed is boiled to a thick porridge with water to which borax has been added,* and wrapped into a cloth. Then the poultice is placed in the so-called cataplasma dish, a square box provided with a tin lining and which is kept constantly hot with an alcohol lamp. The latter method is well adapted for hospital as well as for home treatment. The patient himself may take the hot poultice from the tin box put near his bed. Instead of the hot poultices we may also, according to my suggestion, use the spongiopilin dipped in hot water. My experience, however, causes me to prefer the hot poultices. [Mineral poultices, many varieties of which have recently been put on the market, may also be occasionally employed.] Besides in ulcus ventriculi, they may also be used in gastralgia, cholelithiasis, nephrolithiasis, flatulent colics, etc. More simple and likewise successful in the latter cases are the well-known rubber bags filled with hot water. On the other hand, the thermophores are too heavy and annoy the gastric region. The application of local heat in some gastric affections (particularly gastric ulcer) receives still more encouragement, because, according to investigations of Fleischer,[28] Penzoldt,[29] and others the emptying of the stomach is hastened through it. According to Puschkin,[30] it is supposed that the hydrochloric acid secretion is also increased through hot poultices, a fact which, of course, would be rather disadvantageous in ulcus ventriculi associated frequently with superacidity. According to Penzoldt, however, the hydrochloric acid secretion occurs only at an earlier stage than normally. In recent time,

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* The addition of borax, which, if I am not mistaken, was first made by Quincke, is intended to cover up the unpleasant odour of the linseed.

[28] Fleischer, Berliner klinische Wochenschrift, 1882, No. 7.

[29] Penzoldt, *l.c.*, S. 11.

[30] Quoted from Riegel, Handbuch der physikalischen Therapie, Leipzig, 1902, Bd. 2, S. 32.

Winternitz[31] recommends a method which has proved itself of value in gastric neuroses as well as in organic digestive affections (gastric ulcer, gastric dilatations). Winternitz applies a rubber tube with running hot water (40° C.) to the cold and moist stimulating abdominal packs (wrapped well in dry towel). This direct supply of local heat deadens, according to Winternitz, the unpleasant chilly sensation of the cold wet cloth enveloping the body, as a stronger nerve-stimulus deadens a less intense one. It is noteworthy that in the cases reported by Winternitz the mere application of heat to the gastric region was not accompanied by any favourable result.

In general, cases of gastric and enteric neuroses are adapted for hydrotherapeutic measures. Special indications are very difficult to give, as the reaction to hydriatic measures varies exceedingly in different individuals. In some patients they act soothingly and as a tonic, in others are exceedingly exciting. Such is particularly the case in the irritative form of neurasthenia, in which hydropathic methods may, under some circumstances, bring about a deterioration of the symptoms. In order to test the reaction of the organism towards hydriatic treatment, I usually try to determine the reaction of the patient in this respect by a preliminary trial (cold rubs, cold showers in the tepid bath, etc.). The effect of this preliminary trial is a useful hint for what is to be expected of a systematic water-cure.

The methodic water-cure can usually be carried out with advantage only in *sanatoria*. In these, as a rule, the other physical methods (massage, electricity) may be suitably combined with hydropathic measures. An essential curative factor is offered also by the regulation of the diet, although just in this respect many institutions are very negligent. Only exceptionally and in milder cases an ambulant treatment may also be accompanied by a favourable result.

The Orthopædic Treatment in Diseases of the Stomach.

The orthopædic treatment in affections of the gastro-enteric canal aims to give relief and support to some displaced organs of the abdominal cavity or to prevent a further dropping of the same. The most imperfect, though frequently used, supporter is the "abdominal bandage" in its various forms and types. This has the great disadvantage of easily moving from where the displaced organ is located. For a long time, therefore, bandages have been constructed which were supposed to support the displaced organ (most frequently the kidney) by means of a pelotte (pad) corresponding to the size of the organ. These bandages, which fulfilled their purposes but very imperfectly, have been dropped entirely in the last few years, and an attempt has been made to support the abdominal walls in their entirety, or, as Landau excellently expresses himself "to construct artificial abdominal walls." The abdominal corset, advanced by Landau and more or less suitably modified by others, serves this purpose best. It consists of a heart-shaped covered tin plate which carries two springs connected with an elastic belt. Quite practical, and applicable for all forms of displacement of abdominal organs, is Bardenheuer's bandage. It consists of two springs enclosing the crests of the ilium, and finding there their support and resting-place. These two springs are connected at the symphysis by a third spring. From this there radiate broad pieces of metal or whale-bone inserted into the linen or leather or rubber parts of the bandage, which are provided at the upper end with a belt.

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[31] Winternitz, Deutsche Medicinal-Zeitung, 1891, No. 38.

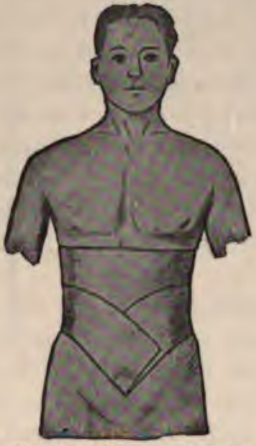

Fig. 49.—Rose's Adhesive Plaster Bandage.

Bandages of similar kind have been suggested in so many varieties that it is impossible to mention even those most frequently used. In every case they ought to be made to measure; it is the duty of the physician to convince himself of the fit and effect of the corset or bandage, and, if necessary, to cause alterations to be made.

These bandages give, if we do not make unreasonable claims, good and palliative service in many cases. The patients feel less troubled and fatigued, are able to perform bodily work to a greater measure, complain less of dragging pains, and do not like to abandon the bandage, the usefulness of which they have proved.

Besides the bandages proper, A. Rose[32] recently used and recommended a bandage of adhesive plaster, which has the advantage of being simple and cheap (Fig. 49). [This bandage does excellent service in many cases.]

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[32] Rose, Deutsche Praxis, 1901, No. 18.

CHAPTER XII.

Lavage of the Stomach, Stomach Pump and Gastric Douche.

Technique of Gastric Lavage.

For the purpose of lavage we employ most preferably a siphon apparatus, by attaching a piece of glass tube to the stomach tube, and connecting it at the other end with a rubber tube to which again a glass [or hard rubber] funnel of not too small a capacity is attached. After introduction of the tube, the elevated funnel is filled with water [while the rubber tube just below the funnel is compressed with a finger], and after the water has flowed off, the funnel is lowered, whereby, through siphon action, possible food-remnants are carried outward. Through elevating and lowering of the funnel the stomach may be cleansed in this manner till clear water runs off.

Besides this simple apparatus, one may, for the purpose of lavage, particularly for self-application, make use of an apparatus suggested by L. Rosenthal and modified by v. Leube. Attach to the tube of an irrigator a Y-shaped tube, the lateral limbs of which are connected with long rubber tubes. If the stopcock of the irrigator is opened, while that of the effluent tube is closed, water flows into the stomach; but if the latter is opened, the stomach-contents run off. For polyclinic purposes I formerly put a 5-litre bottle upon the stand and connected the effluent opening, according to v. Leube and Rosenthal, with a triangle, one limb of which was connected with a rubber tube which carried a glass funnel. Hereby the possibility was given to observe the effluent stomach contents at any moment. A similar apparatus is suggested by Litten[1] (Fig. 50).

But neither Rosenthal's nor Litten's apparatus has been generally adopted, because these require a second person for filling of the irrigator.

The liquid used for lavage must be lukewarm (about 30° C.) [There are cases, in which hot (40° C.) and cold (15° C.) water may be of great service]. The quantity of the fluid necessary for cleansing the stomach depends on the type of the gastric affection; sometimes five or six litres of water, or even more, are required for complete cleansing. We should take care that the effluence corresponds to the affluence, and therefore we should not pour the lavage fluid into the stomach all at once, but only gradually [in measured quantities (a funnelful)]. A stoppage of the effluence may depend on the fact that either the fenestra of the tube is not immersed, or that it is obstructed by food particles. In the former case further affluence immediately restores the interrupted effluence;* in the latter case air

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[1] Litten, Therapeutische Monatshefte, 1893, S. 255.

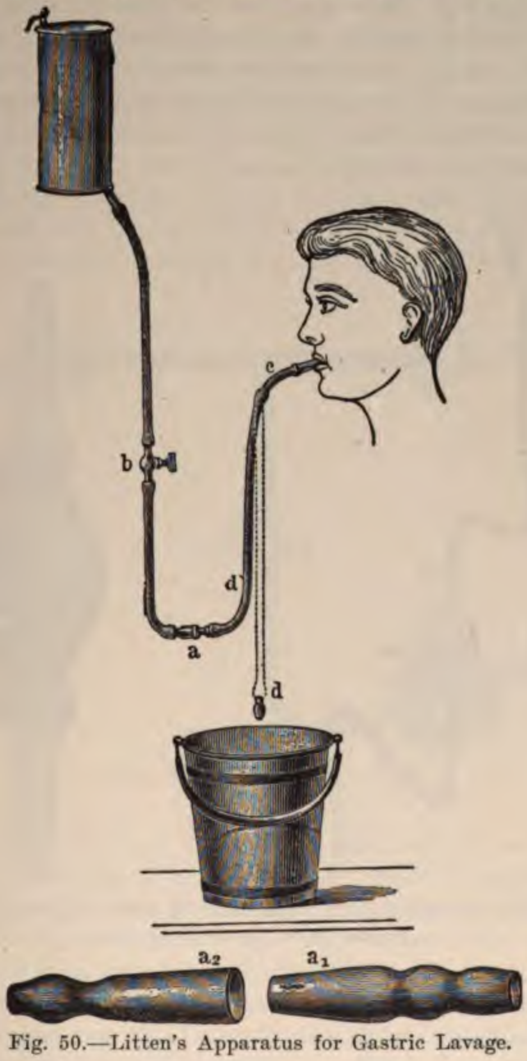

Fig. 50.—Litten's Apparatus for Gastric Lavage.

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* [At the time of affluence the stomach tube should reach only to the cardia or very little below, and before effluence is desired the tube is pushed farther down; how far depends on circumstances.]

should be cautiously driven through the tube by means of a double bulb, whereby the obstructing substance is expelled with a distinct noise.

Quite similar is the apparatus suggested by Friedlieb[2] (Fig. 51) and H. Strauss[3] (Fig. 52). The apparatus of Strauss has a permanent double bulb inserted between stomach sound and rubber tube by means of a T-shaped piece. Through this it becomes somewhat unhandy, and I do not see the advantage which the apparatus is said to possess compared with the application of a double bulb, if the same is needed. The instrumental apparatus cannot be made simple enough.

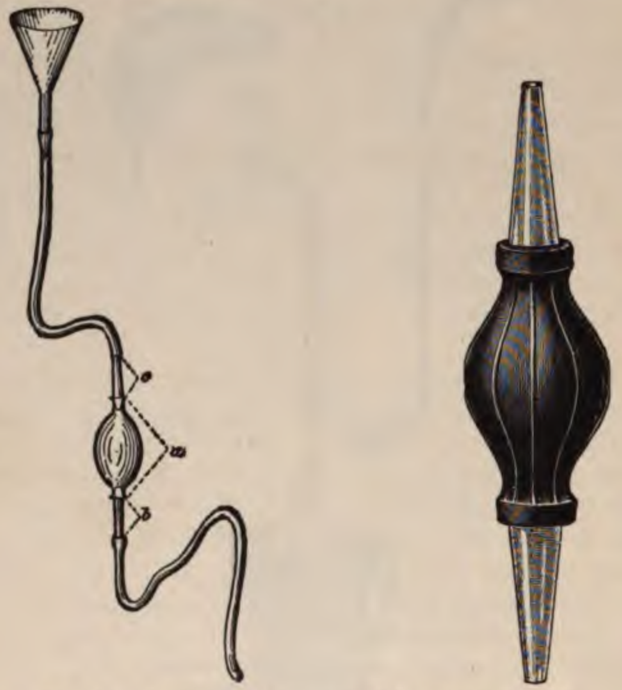

Fig. 51.—Friedlieb's Apparatus for Gastric Lavage.
a, Rubber bulb. *b*, *c*, Glass tube.

As soon as the flow of clear wash-water out of the stomach ceases, I remove the rubber tube and funnel; the fluid still remaining in the stomach is expelled by expression. The quantity remaining in the dilated stomach is considerable, and its removal is of great importance. [Instead of the method of expression in such cases, I often prefer removal by aspiration, as in the examination of the stomach-contents; the alternate use of hot and cold water for lavage purposes is of great service].

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[2] Friedlieb, Deutsche medicinische Wochenschrift, 1893, No. 51.

[3] H. Strauss, Therapeutische Monatshefte, 1895, Märzheft.

According to Fleiner's[4] suggestion, we may, after lavage of the stomach while the patient is sitting, tell him to lie down, and in this position we may continue to wash the stomach with another litre or more of water, whereby frequently considerable remnants are brought forth; this procedure presupposes a certain practice of the patient. Advantageous also is the direction given by Fleiner to have the patient while sitting, or still better while lying, make shaking movements; through these the remnants possibly remaining in the stomach are more thoroughly mixed and diluted.

To the wash-fluid may be added salts or mineral waters or their constituents, then antiseptic remedies, or finally astringent substances. Among the first-mentioned the most frequent are table salt (1 per cent), sodium carbonate or bicarbonate (2 to 5 per cent.), [milk of magnesia], Carlsbad salts, Carlsbad waters, waters of Ems, Vichy, Kissingen, Neuenahr, etc., either in their original form, or, what may be preferable, in the form of artificial mineral salts (Sandow, [Wyeth and others]).

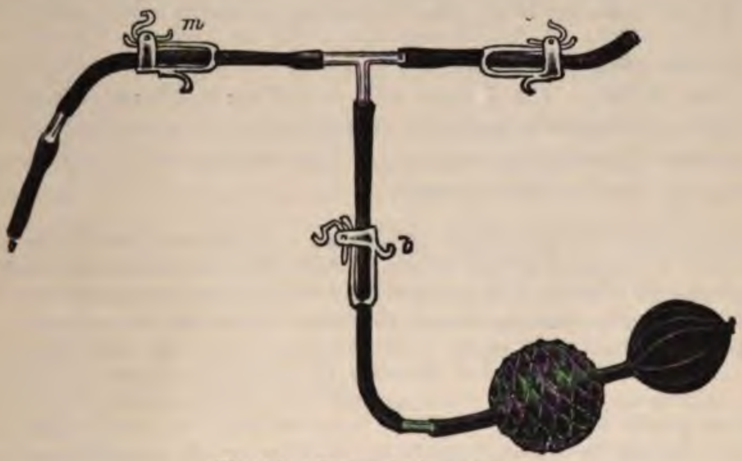

Fig. 52.—Strauss's Apparatus.

As antifermentative additions to the wash-water, almost all antiseptics, except the poisonous (carbolic acid, corrosive sublimate), have been suggested, particularly potassium permanganate, thymol (one half per cent.), resorcin* (2 to 3 per cent.), salicylic acid (3 to 1000), sodium salicylate (0.5 to 1 per cent.), sodium benzoate (1 to 3 per cent.), boric acid (3 per cent.), chinosol (1 per cent.), ichthyol (1 to 1.5 per cent.), lysol ($^1/_4$ to $^1/_2$ per cent.), [glyco-thymoline], etc. Dujardin-Beaumetz recommended also hydrogen sulphide water, without having, as it seems, found any followers. M. Rosenthal recommended lavage with water containing amyl nitrite (3 or 4 drops of amyl nitrite in 500 cubic centimetres water). I use exclusively boric acid in 3 per cent. solution, sodium salicylate in 2 per cent. solution, or salicylic acid solution (1 to 300); usually I alternate every week in the single case.

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* The same must be chemically pure (resorcinum resublimatum).

[4] Fleiner, Volkmann's Sammlung klin. Vorträge, 1894, No. 103.

The practical value of these additions seems to me the less important the larger my experience becomes. In order to effect a perfect removal of the germs of fermentation, the lavage with antiseptic solutions is certainly not sufficient, and for the mechanical removal sterile water will probably suffice. Only in putrid conditions (ulcerating carcinoma) are antiseptic solutions (lysol, creolin, ichthyol, etc.) indicated, and, according to my observations, they are useful to a certain extent.

Among astringent substances we have to consider bismuth subnitrate and silver nitrate. The former acts, according to Fleiner,[5] in irritative conditions of the stomach, due to chronic ulcers, in ulcerating carcinoma, in hæmorrhagic erosions, and gastric hæmorrhages which are discovered on lavage or develop during lavage.

The technique is, according to Kussmaul's and Fleiner's directions, as follows: First, early in the morning, the stomach is thoroughly cleaned through lavage; then a suspension of bismuth (10 to 20 grammes in 200 cubic centimetres of water) is introduced into the stomach through the tube. The patient is now told to lie on his right side (if the seat of the ulceration is near or at the pylorus), or on his back (if ulceration is situated at the lesser curvature), and the tube is shut off by means of a clamp. After five or ten minutes the bismuth precipitates so completely upon the gastric walls, that the water runs off clear. In restless patients the stomach-tube may be removed immediately after the introduction of the fluid. The administration should be applied, in the beginning, every day; later, every other day. [In certain cases I administered even 30 grammes of bismuth in about 75 cubic centimetres of warm water. Symptoms of intoxications I have never observed. It is absolutely necessary to use a chemically pure bismuth, the volume of which must be relatively large, and therefore light, as otherwise the suspension does not become uniform, and, on the other hand, the bismuth will easily cake. (Merck, Rosengarten). Quite recently I used a preparation called milk of bismuth, which seems to be of good service; though it requires rather large quantities]. Intoxication was not observed by Fleiner in spite of long continued application of bismuth. Various authors, Laquer,[6] Kuck,[7] Manasse,[8] Lissauer,[9] Maybaum,[10] Elsner,[11] Starck,[12] and others, have made the attempt to replace bismuth subnitrate through bismutose, a combination of bismuth and albumen with an amount of 21 per cent. bismuth. The results correspond, of course, to those of bismuth; the remedy is praised for its good tolerance and absence of symptoms of intoxication.

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[5] Fleiner, Verhandl. d. XII Kongr. f. innere Medicin, Wiesbaden, 1893.

[6] Laquer, Therapie der Gegenwart, 1901, Juliheft.

[7] Kuck, *ibid.*, 1901, Novemberheft.

[8] Manasse, Therapeutische Monatshefte, Januar, 1902.

[9] Lissauer, Deutsche medicinische Wochenschrift, 1902, No. 33.

[10] Maybaum, Fortschritte der Medicin, 1902, No. 26.

Occasionally I employ *silver nitrate* with good results in myasthenia of the stomach associated with supersecretion, making irrigations with one litre of silver nitrate solution (1 to 1000) after previous lavage, and in the beginning every day, later several times a week. [I never use silver nitrate oftener than every other day; on the alternating days I employ solution of alumnol. In spite of many hundreds of applications, I never saw toxic manifestations.]

After a brief action the fluid is removed. If marked burning is felt, patient may drink a diluted salt solution (1 to 200); but as a rule it is not necessary.* Instead of silver nitrate solution, the weaker protargol, in 0.5 to 1 per cent. solution, may also be employed [I have tried argyrol, but without special benefit].

Of importance is the question, how often and at what time of the day should lavage be performed? As regards the first question, I am of the opinion that when a special indication for lavage is at all existing, it should be undertaken daily. Only in conditions of grave weakness may lavage be carried out in intervals of one or two days. To wash out more than once a day is probably seldom necessary. [Bettman and others recommend two lavages in certain dilatations, one in the morning, and one in the evening; at the same time they recommend only two meals, and that large ones, to be taken some time after the lavage.]

As regards the time of the day, we have to consider early in the morning before breakfast and late in the evening at bed-time. Either time has its advantages and drawbacks. The advantage of the evening lavage, which Riegel[13] and his disciples particularly favour, consists, as the said author justly emphasises, in the fact that the stomach obtains a longer rest, which is certainly important for the regaining of the normal tonus. I see another advantage in the evening evacuation, namely, that thereby the opportunity for fermentative and putrefactive processes in the stomach is removed for twelve hours. On the other hand, the evening lavage has the drawback that the organism is deprived of more or less large quantities of ingesta through the washing-out. This drawback does not exist at the morning lavage, because the remnants of chyme, not propelled into the intestine or not absorbed by the stomach during the night, can hardly be of any use for nutrition.. The morning lavage also furnishes an important diagnostic or prognostic sign. We can readily convince ourselves how great the impairment of the peristalsis is, and whether it increases or decreases in the course of the treatment. Finally, we have to emphasise the very important factor that the evening lavage, applied immediately before bedtime, is associated in practice with great, and occasionally insuperable difficulties.* Therefore I usually wash out the stomach in the morning, and select evening lavage only in special cases, as, for instance, in cases of frequent pain or vomiting during the night.

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* After having observed a case of argyria after abundant application of silver irrigations, I recommend great caution and an application not to be continued for too long a time. [The silver nitrate solution may remain in the stomach for about two or three minutes; after removal of the original solution, it is necessary to wash the stomach with water as long as the fluid still appears milky (silver chloride); usually two or three funnels of water are sufficient for this purpose. If we are still in doubt whether silver nitrate might have been left, it is advisable to wash out with a sodium chloride solution].

[11] Elsner, Archiv für Verdauungskrankheiten, 1902, S. 638.

[12] Starck, Münchener medicinische Wochenschrift, 1902, No. 47.

[13] Riegel, Die Erkrankungen des Magens, I Theil, S. 293, Wien, 1896; also Handbuch der physik. Therapie, Leipzig, 1901-1902, Bd. 2, S. 299 u. f.

Riegel and others assume that with evening lavage the stomach, getting longer relief, becomes better enabled to overcome its task. This view, however, seems to be more theoretic than practically proved. The essential argument for the evening lavage rests, in my opinion, principally in the fact whether the nocturnal troubles are relieved or removed. In this factor alone, unless the favourable influence upon the motility is unequivocally established, I see the indication for the selection of the evening time. Moreover, the whole question is not of such a great importance as Riegel assumes. The essential point is whether the symptoms of stagnation can be permanently removed at all through internal methods; if otherwise, we have to resort to surgical intervention.

As *stomach pumps,* various more or less useful kinds of apparatus are employed, which all depend on the same principle, namely, to produce pumps with a double valve, through which exhausting and inflating action is obtained in a simple manner. A very useful apparatus is that constructed by Wymann and manufactured by C. Möcke, of Leipsic.

Personally, I never employed the stomach pump clinically, and regard it for reasons already mentioned (page 137) as a dispensable instrument. The drawback formerly assigned to the siphon apparatus (v. Leube), namely, that only liquid substances could leave the stomach, is entirely ephemeral, as it is only a matter of size of the lumen of the tube and of its openings. That quite large food particles may be removed by means of the siphon, I have often had the opportunity of observing.

In brief, I am of the opinion that no result in cleansing the stomach can be obtained by means of the stomach pump which could not also be obtained by siphoning. The stomach pump, therefore, deserves its place at the side of its twin sister, the clyster syringe.*

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* [Evening lavage is more adapted for hospital and sanatorium practice.]

That we do not desire to belittle the epoch-making invention of Kussmaul can be seen from the introductory remarks of this work: *not in the mechanical carrying out of the idea, but in the idea itself, we see the immortal merit of this ingenious clinician.*

Indications and Contra-Indications of the Lavage of the Stomach.

When we omit those washings-out of the stomach which are applied in cases of poisoning, ileus, cholera infantum, cholelithiasis, jaundice, threatening rupture of the stomach, etc., as not belonging to our subject, we may give the indications of lavage as follows:—

Gastric lavage is indicated (1) when there exist mechanical obstacles to the propulsion of the chyme in the gastro-enteric canal, which cause abnormal decomposition; (2) when alien substances are admitted with the stomach-contents, which gradually destroy digestive action.

As may be seen from these indications, lavage of the stomach *is applied far oftener in practice than it is indicated.* The more, however, we limit its application, the less we will discredit this curative measure, so effective when used under the right indication.

To the mechanical obstacles belong above all, as is well known, the gastrectasis due to cicatrised stenosis at the pylorus and duodenum, then the impairment of the motor function due to organic affections of the gastric mucosa (carcinoma, atrophy of the mucous membrane, amyloid, corrosion of the mucosa through acids and caustic alkalies, etc.), but only *in so far as they lead to retention of food.* Hereby are also satisfactorily limited the types and degrees of the dilatation in which, in my opinion, lavage should be performed. On the ground of many bad results, I am of the opinion that simple atony, in which we have to deal not with a direct obstacle, but merely with a retarded peristalsis, does not offer a fit object for lavage. On the contrary, we may, if not careful that evacuation of the lavage water occurs, frequently observe deteriorations. We have but to remember that the washing-out by no means attacks the cause or acts symptomatically to any special extent; indeed, it involves the danger of superdistension of the gastric wall, an undesirable factor.

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* [I may remark here, that the clyster syringe, modified, has of late, regained to a certain extent, its place in the armamentory of the physician. As well known, oil is a destroyer of *soft* rubber, and of late years the *hard rubber* clyster syringe (perhaps with or without an attachment of a soft rubber rectal tube, which could be used at least a few times) has frequently been of use in the application of the oil enema].

As regards the effect of lavage in gastrectasis, we must distinguish at what stage it is applied. If a severe cicatricial pyloric stenosis is present, and resorption and urine secretion are greatly reduced, the effect at its best is but palliative. The patients are always confined to the use of the stomach tube.

It is quite different, however, with the more recent and milder cases of the said type (so-called relative pyloric stenosis), a large number of which I have seen cured. In such cases the fasting stomach either contains nothing or but a slight amount of gastric secretion after the course of treatment, the patients eat and tolerate the usual food without special precautionary measures, diuresis and bowel movements become normal, the patients gain in weight of the body. Probably we have to deal in these cases with a compensatory hypertrophy of the muscles which enables the stomach to expel the food again in the usual manner. There is no doubt that through excessive overloading of the stomach disturbances of the motor function may occur anew, but nevertheless in my opinion the fact remains unrefutable that cases of ectasia not advanced too far may be cured by suitable treatment. To this class evidently belong also the cases described by Kussmaul[14] in his fundamental treatise.

Less clear and urgent is the indication of gastric lavage on accumulations of heterogenous substances. To them belong mucus, gastric juice, bile, blood, and blood and pus. In all these cases, to be sure, the stomach can be freed quickest of these admixtures by means of lavage. This procedure, however, is not always necessary, as, for instance, accumulation of mucus may be removed just as well by drinking warm solutions of salt or soda. Likewise pain due to excessive acid production may be removed by administration of alkalies just as well as by lavage. Considerable accumulations of bile are preferably removed by lavage. Slight hæmorrhages are best left to themselves; in severer ones Ewald[15] and Minkowski[16] recently recommended irrigations with ice-water, but only as a last resort. Only more extended experiences can decide on their value.

Gastric lavage is *contra-indicated* in all cases where the introduction of the tube is also contra-indicated. Particularly *fresh* gastric hæmorrhages, even when a cause for lavage (for instance, in pyloric stenosis with recurrent ulcer), as well as cancers with ink-like stomach-contents, furnish, according to my experience, a decided contra-indication of lavage. If in the latter an evacuation must be performed, we ought to content ourselves with expression.

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[14] Kussmaul, Deutsches Archiv für klin. Medicin, 1869, Bd. 6, S. 455.

[15] Ewald, Verhandlungen des Kongresses für innere Medicin, 1902.

[16] Minkowski, 67 Versammlung deutscher Naturforscher und Aerzte, ref. Centralblatt für innere Medicin, 1895, No. 40.

I wish to protest most decidedly against the aimless experimenting with gastric lavage, which unfortunately is not condemned enough by clinicians of even the widest reputation; there are actually but rare cases in which "accidentally" gastric lavage is accompanied by results. In regard to some, I might suggest that the result was due only to the introduction of the stomach tube; for instance, in several cases of habitual vomiting (irritable stomach) I brought about a cure merely by allowing the stomach tube to remain in the stomach for some length of time. In one case almost all nervina had been internally tried without any effect. Therefore, I made use of lavage, but for the above reason I believe that no essential value ought to be ascribed to the introduction of water. The course justified this conception, because only the application of the tube brought about permanent cure. Accordingly, I consider also the results which Bendersky[17] recently obtained by means of gastric lavage in nervous vomiting, as purely suggestive ones.

The Gastric Douche.

By *gastric douche* is understood the irrigation of the stomach with water under high pressure, for the purpose of influencing therapeutically the gastric mucous membrane. This method was first practised in Kussmaul's clinic, and was described by Malbranc.[18] It is supposed to incite the circulation and to mechanically increase the peristalsis by stimulation of the vasomotor nerves of the stomach. Malbranc observed its favourable effects particularly in obstinate gastralgias. He made use of warm soda solution (38° C.) as an irrigation-fluid. In several cases of gastric neuroses, I employed the method by introducing a sound perforated with many small openings the size of the head of a pin, and through it let water flow into the stomach. This irrigates the walls of the stomach with a fountain-like stream. As far as the technique is concerned, very many devices have been suggested, all of which are based on the same fundamental idea. The apparatus of Einhorn (Fig. 53) answers all purposes.

This consists in a stomach-tube of about 60 centimetres in length and 1 centimetre in thickness, on the end of which an oval capsule-shaped piece of hard rubber has been attached (1). This is provided with numerous small perforations, and has a large, round hole at the end. In this capsule, which may be screwed off from the tube proper, there is a small aluminum globe, which may play about inside the capsule, but fully closes the lower opening after the tube has been introduced. Two crossed pins in the upper part of the capsule (2) prevent the globe from entering the stomach-tube. After the apparatus has been attached to an irrigator furnished with a tube by which the water may flow off (or to a siphon-apparatus), the water will flow in a fountain-like spray through the small holes, while the sphere prevents the flow through the large opening. On the other hand, when the entrance tube is closed, the returning water will raise the ball upwards, so that it can flow readily through the lower opening (and also through the small holes). It is well, on introducing the tube, not to bring it much below the cardia; on the other hand, it should be pushed in 10 to 15 centimetres further on the return flow.

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[17] Bendersky, 13 internationaler Kongress in Paris, 1900.

[18] Malbranc, Berliner klinische Wochenschrift, 1878, No. 4.

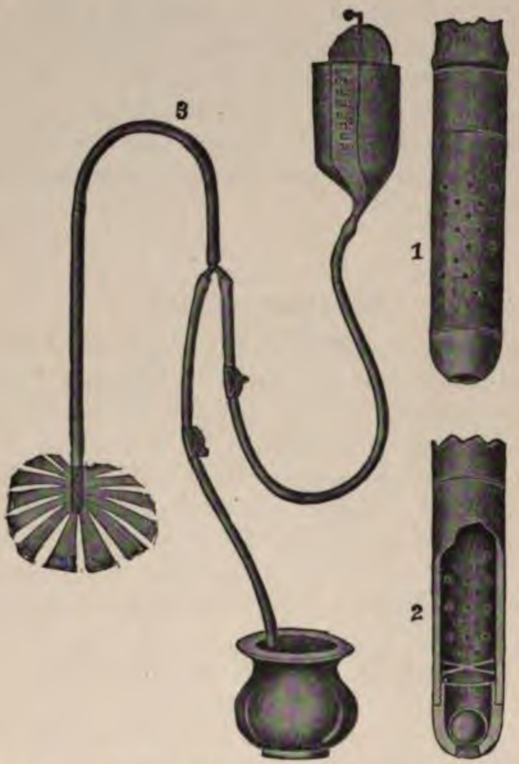

Fig. 53.—Einhorn's Apparatus for Gastric Douche.

It is self-evident that the gastric douche should be employed only on an empty stomach. The liquid for irrigation may have medicinal substances added, as, for example, common salt solution, silver nitrate, chloroform water, soda, Carlsbad salts, etc. According to the exigencies of the case, these may be used to exert a sedative or a stimulating effect on the mucous membrane.

My own experience with the gastric douche does not lead me to employ the method very frequently, and Penzoldt[19] also expresses himself very sceptically concerning it. Of late years we have heard very little of the results of gastric douches, and it seems already to have disappeared from the order of the day. They may have, however, an occasional use as a suggestive remedy in neurasthenics.

Einhorn has also constructed (Fig. 54) a so-called powder-blower for local treatment by means of soluble or insoluble substances. This consists of an ordinary stomach tube, the one end of which is attached by means of a hard-rubber piece to a bulb *B* provided with a valve, and the other end leads to a hard-rubber piece *C*, to which a capsule *D* is screwed. The endpiece *C* is hollow, carries several small openings for letting the air pass through, and is provided with a screw-thread for the additional piece. The capsule *D*, which has many perforations and which comes in three different sizes (D, E, and F, 3, 3½, and 4 centimetres in length) is filled with the necessary amount of powder and is screwed on the hard-rubber piece. It is self-evident that the dusting of the stomach with powder is to be undertaken only when the stomach is empty. Whether this apparatus is of material advantage remains to be seen.

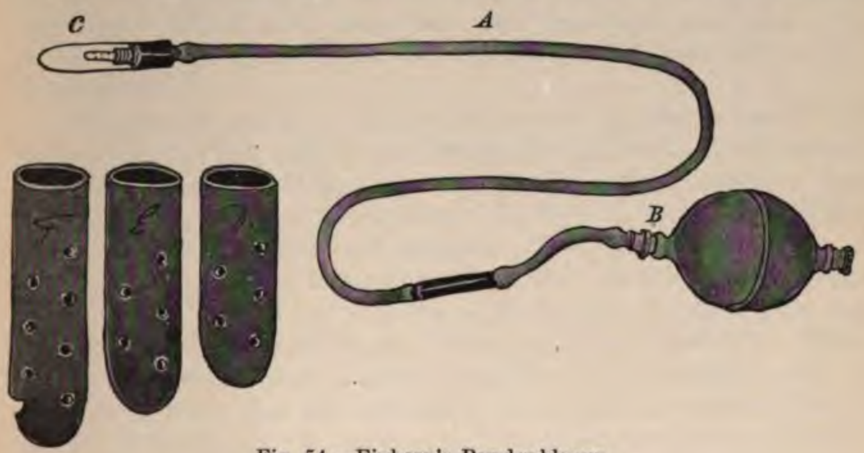

Fig. 54.—Einhorn's Powder-blower.

Expression of the Gastric Contents.

We can use the expression of the gastric contents for therapeutic as well as for diagnostic purposes. For example, in gastrochlyorrhœa and also in slight fermentations in the stomach, but especially in very weak patients or in individuals sensitive to the use of the sound, it may even be preferred to lavage of the stomach. Also for partial evacuation of the stomach-contents in severe pain, for example, during the night, auto-expression suffices to a high degree, and it is not yet established whether in such cases it accomplishes less than the more circumstantial washing out of the stomach.

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[19] Penzoldt, Allgemeine Behandlung der Magen und Darmkrankheiten in: Penzoldt-Stintzing's Handbuch, 1896, Bd. 4, S. 299.

CHAPTER XIII.

Employment of Acids and Alkalies.

The Acids.

In chronic diseases of the stomach we employ the acids, especially the mineral acids, and hydrochloric acid is the first among all of these. Hydrochloric acid brings about the most favourable combination as to digestion; sulphuric acid, nitric acid, and phosphoric acid have a far smaller effect of separating pepsine and lab-ferment from the secretion of the glands.

What is the action of the hydrochloric acid in the stomach? The views concerning this question are divided. On the ground of experiments on healthy persons, to whom he gave hydrochloric acid in ascending doses simultaneously with the test-breakfast, du Mesnil[1] believes that hydrochloric acid increases the acid secretion. This conclusion, however, as Riegel correctly remarks, is not to be drawn from du Mesnil's own figures, which, in fact, without and with the administration of hydrochloric acid, show very considerable variations. More recent investigation by Schüle[2] showed no influence at all, even with large doses of hydrochloric acid. It is far more likely that the hydrochloric acid exerts an influence on the motility of the stomach; to this end, perhaps, speak experiments of Eichenberg,[3] who observed an acceleration of digestion of from 20 to 25 minutes under the administration of hydrochloric acid. The uncertainty of our knowledge concerning its physiologic action is also reflected in the treatment. Shall we give large or small doses, shall these be given before or after eating, and shall repeated small doses or a single large dose be administered? As many questions, just as many different answers. Ewald[4] advises the administration of doses as great as possible (90 to 100 drops) three or four times, at intervals of fifteen minutes; while Cahn[5] considers these doses too large. Other authors also, for example v. Leube, are satisfied with very much smaller doses. Riegel[6] is of the opinion that we must determine the dose in the individual cases. I have seen no worse results follow small doses administered at each of the principal meals than after the use of the large doses, but I am of the opinion that the matter has been by no means cleared. One can expect, for instance, that with a greater amount of hydrochloric acid more albumen is changed to acid-albumen, and thus, though perhaps only incompletely, is digested. On the other hand, we know that in achylia of highest degree, in which light bread and meat can be demonstrated to leave the stomach as they enter it, even the slightest objective or subjective disturbance may be absent. But when with the use of hydrochloric acid subjective disturbances disappear, is this really only the effect of the hydrochloric acid therapy, or not rather due to the care taken with the diet? We may see that the matter is not so simple.

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[1] du Mesnil, Deutsche medicinische Wochenschrift, 1892, No. 49.

[2] Schüle, Habilitationschrift, Berlin, 1895, P. 69 and further.

[3] Eichenberg, citad by Penzoldt. Allgemeine Behandlung der Magen- und Darmkrankheiten, in Penzoldt-Stintzing's Handbuch der Therapie, 1896, IV, P. 273.

[4] Ewald, Berliner klinische Wochenschrift, 1886, No. 4.

[5] Cahn, Zeitschrift für klinische Medicin, 1887, Bd. 12, P. 42.

On the whole, the following principles have proved of value to myself. When it is desired to get the *digestive* action, I give the hydrochloric acid (acid. hydrochlorat. offic.) immediately or shortly (15 to 30 minutes) after ingestion of food, in doses of 8 to 10 drops in a wineglassful of water, and that once after small meals and two or three times in repetition after larger meals. In order to get the stimulating or antizymotic action I prefer administering the hydrochloric acid at other times than during meal times, as the intended action is best brought about then. In such cases I give the hydrochloric acid on an empty stomach in the morning, and in the evening before going to bed, in equal doses. In order to protect the teeth, it is better to direct the acid to be taken through a glass tube.

The question as to whether we can bring back to normal the lost secretion of the glands of the stomach by means of hydrochloric acid *alone* is also answered differently. Jaworski[7] is of the opinion that the acidity in general is lowered after a long administration of hydrochloric acid. On the other hand, Riegel[8] could, after the use of 1.5 grammes hydrochloric acid for two weeks, repeatedly demonstrate free hydrochloric acid in the fasting stomach-contents of a patient who had previously had no free hydrochloric acid for months. Reichmann and Mintz[9] also increased the secretion of gastric juice, that is, of the hydrochloric acid, by the use of the acid in several cases of diminished secretion. L. Wolff,[10] on the other hand, could not demonstrate on any of the subjects of his experiments a change of chemistry after a week's employment of large doses of hydrochloric acid. My own experience has convinced me that the effect of the continuous therapy by hydrochloric acid varies very much; while in one group of affections free hydrochloric acid returns, in another group colour-reactions do not appear even with the use of hydrochloric acid continued for years; and finally, in a third group, traces of hydrochloric acid are produced, and traces they remain. This is quite natural when we consider, as I have repeatedly pointed out, that the lack of hydrochloric acid may originate from several causes. That in certain cases (neuroses, stagnation-catarrh) deficiency of hydrochloric acid is permanently improved less by the administration of hydrochloric acid than by suitable diet, as well as by the betterment of the external circumstances of the patient (change of air, sea-baths, etc.), is for me an incontrovertible fact.

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[6] Riegel, Zeitschrift für klinische Medicin, 1886, Bd. 11, P. 213.

[7] Jaworski, Deutsche medicinische Wochenschrift, 1887, No. 36-38.

[8] Riegel, Deutsches Archiv für klinische Medicin, 1885, Bd. 36.

[9] Reichmann and Mintz, Wiener klinische Wochenschrift, 1892, No. 25.

[10] Wolff, Zeitschrift für klinische Medicin, 1889, Bd. 16, P. 224.

In almost all large pharmacies pills or dragées of hydrochloric acid, pepsine, and aromatic vegetable powders are for sale. I consider this kind of administration of the acid mere tomfoolery and am of the opinion that any beneficial result induced is due to suggestion. If we are convinced, as the author is, how few are the cases of secretory disturbances in which the hydrochloric acid thus given succeeds in acting, we would soon retract the view that we can obtain an influence on digestion worthy the name by the use of two or three pills. As far as the pepsine is concerned, it will be considered later on.

As contra-indications to the use of hydrochloric acid, as it seems almost superfluous to mention, must be considered all forms of abnormal increased secretion of the glands. There is also said to be, according to Talma,[11] a kind of hyperæsthesia against hydrochloric acid, although, as Riegel[12] correctly says, this may really be a condition of hyperchlorhydria.

Animal Gastric Juice (Gastérine).

The gastric juice of dogs, recovered according to the principle of Pawlow's gastric fistula, has been introduced into therapy under the name of "gastérine," and tested by French investigators (Frémont,[13] Mathieu and Laboulais,[14] and others). Especially good results have been reported as to this remedy in hypochylia. In fact, Huchard[15] goes so far as to ascribe to gastérine in this trouble the same effect as to digitalis in cardiac affections. Finkelstein,[16] also, praises the action of the natural gastric juice of the dog in catarrh of the stomach, in anæmia, in typhus abdominalis, and even in carcinoma ventriculi. Of German physicians, to my knowledge, P. Mayer[17] alone has employed this gastric juice with pregnant results, in a case of apepsia gastrica with diarrhœa.

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[11] Talma, Zeitschrift für klinische Medicin, 1884, Bd. 8, P. 407.

[12] Riegel, *l.c.*

[13] Frémont, Bulletin général de thérapeut., 1898.

[14] Mathieu et Laboulais, Société méd. des Hôpitaux, 1901.

[15] Huchard, Bull. de la soc. de thérapent, 1899, No. 16-17; 1900, No. 23-24.

I have two observations at my disposal. Both were cases of apepsia gastrica with obstinate diarrhœa. In one case no results at all followed the use of gastérine; on the second the subjective influence on the condition was decidedly good, so that the afflicted patient repeatedly used it. The effect upon gastric digestion was not established, for after the omission of gastérine the gastric contents showed just the same composition as previously.

In any case it can exercise only a symptomatic and, in this sense, perhaps, valuable effect; an objective and lasting impression upon the disturbed gastric digestion is hardly to be expected. The dose of gastérine is one, two, or three tablespoonfuls every three hours, with or immediately after meals.

[Several years ago I had at the same time two patients under my observation, the one suffering from nervous gastrochylorrhœa (free HCl up to 125), the other with a marked atonic dilatation of the stomach and chronic gastritis (abuse of beer—quantities of fluid, as much as 10 to 12 litres a day) and with a great loss of hydrochloric acid. The one patient furnished as much as 500 to 1000 cubic centimetres of the very strong hydrochloric fluid every day. After having employed in the second patient the usual hydrochloric acid in even large doses without any special effect, I decided to try the human hydrochloric fluid of the first patient. I filtered the contents, so that I obtained an absolutely clear fluid, added a few drops of chloroform (or also 0.5 per cent. of carbolic acid) and gave it to the patient suffering from loss of hydrochloric acid, with the direction to take one tablespoonful of the fluid before and after meals three times a day. Numerous examinations of stomach-contents of the patient, whose condition had lasted for about nine years, showed no HCl after a Boas-Ewald breakfast on April 29, 1900; nor on May 5th, after a Riegel meal, nor May 13th, after a Riegel meal, nor May 20th, May 27th, June 17th. There was never lactic acid; total acidity was always very low. On June 24th, slight bluish reaction on Congo; after Ewald breakfast no free HCl on July 15th; but Congo reaction again August 22d, after Riegel meal; no free HCl on September 7th, after Ewald breakfast. Patient, who of course did not know the origin of the medicine, felt decidedly better after its administration than after the commercial hydrochloric acid. Of course, patient had been put also on a diet, and lavage of the stomach was used. Lavage often brought forth food eaten some days previously. Patient has been in good condition since.

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[16] Finkelstein, Wratsch, 1800, No. 123.

[17] P. Mayer, Zeitschrift für diätetische und physikalische Therapie, 1900, Bd. 4, P. 220.

We must not forget that the hydrochloric fluid secreted in the stomach is different from the hydrochloric acid bought in the drug store, as readily can be proved by the calcium oxide test.

A new preparation, called "gastron," has been manufactured in America, but not yet brought on the market for sale. The product is made directly from the gastric cells; it is claimed to have the acid content of normal gastric juice, 0.2 per cent., with glycerine, 20 per cent. Examination shows no free HCl, but an acidity of about 100 (combined HCl); on addition of a very few drops of a very dilute hydrochloric acid solution *free* hydrochloric acid can be demonstrated. The peptonising activity is excellent. The lab-ferment does not appear to be very effective in the preparation. "Gastron" deserves further and thorough experiments.]

The Alkalies.

The common employment of the alkalies in diseases of the intestinal tract, and their great usefulness when indications are correct, make it incumbent on us to consider exactly the manner of their administration and their physiologic action, although the latter is known to us only in part.

As a rule, the alkalies are used either alone or (mostly) in combination with other salts or vegetable powders; for the most part, however, the basis of the prescription is sodium bicarbonate (potassium bicarbonate), or (more rarely) sodium carbonate, calcium carbonate or the preparations of magnesia. The combination of these agents with other more or less indifferent additions forms the basis of the preparations for the stomach which are sold under names high-sounding and full of promise (among others, the well-known *Barella's stomach powder** which, besides sodium bicarbonate, very considerately also contains pepsine).

Carlsbad salts, partly as the natural product of the thermal water, and partly as artificial mixture, have played for a long time such an important rôle as an antacid, as well as a stomach remedy in general, that it becomes indispensable for us to consider briefly the value of this drug and the indications for its use.

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* It is a sad sign of our times, as I have convinced myself, that even physicians prescribe this ridiculous preparation for their patients and recommend it to them.

From Carlsbad thermal water two products are obtained, the composition of which differs entirely, and they consequently show a different pharmacodynamic action: *the natural Carlsbad Sprudel salts (crystallised), and the natural Carlsbad Sprudel salts (pulverised).*

The first-named salt, first made to a greater extent by the celebrated Carlsbad bath-physician, David Becher (1764), consists, as the following analysis will show, principally of sodium sulphate, whereas, in contrast to the composition of the thermal water, sodium carbonate and common salt are present only in a very slight amount. The crystallised Sprudel salt shows the following composition:—

Sodium sulphate	37.695 per cent.
Sodium chloride	0.397 per cent.
Sodium carbonate	5.997 per cent.
Potassium sulphate	Traces
Water of crystallisation	55.520 per cent.

In Sprudel salts free of water of crystallisation, Harnack found 99.33 per cent. of sodium sulphate, 0.45 of sodium carbonate, and 0.076 per cent. of sodium chloride.

Only since 1882 pulverised Sprudel salts have been on the market, which, made according to the directions of Prof. E. Ludwig, of Vienna, contains, with the exception of the earthy carbonates, the iron, manganese and aluminum oxides, and the silicic acid, all the constituents of the Sprudel water that are soluble in water, and that in exactly the same quantitative proportions as in the thermal water.

It is obtained through boiling of the Sprudel water, removal of the occurring precipitate (sinters), filtration, evaporation of the filtrate until a salt mass containing still some per cent. of water is obtained, and saturation of this with carbonic acid. The latter is done in order to reconvert the bicarbonate of soda decomposed during the concentration of the water, back again to bicarbonate. Finally, the powder is freed from water in dry air.

According to Prof. E. Ludwig, the salt thus obtained has the following composition:—

Sodium sulphate	41.62 per cent.
Potassium sulphate	3.31 per cent.
Sodium bicarbonate	36.11 per cent.
Sodium chloride	18.19 per cent.
Lithium carbonate	0.2 per cent.
Sodium borate	0.03 per cent.
Water	0.44 per cent.

Almost entirely analogous to the natural Sprudel salts is the so-called "artificial Carlsbad salts" (Sal Carolinense factitium. Pharm. Germ.). It consists of 44 parts of dry sodium sulphate, 2 parts of potassium sulphate, 18 parts sodium chloride, and 36 parts of sodium bicarbonate. If we ignore the lithium and sodium borate, which are certainly indifferent pharmacologically, there is no reason to replace the artificial salt by the considerably more expensive natural salts. Nevertheless, the natural salts taste better, and on this account are commonly preferred to the artificial.

According to Jaworski, the minimal dose of (pulverised) Carlsbad Sprudel salts is 3 grammes (1 teaspoonful); the medium dose, 10 grammes (2 teaspoonfuls); and the maximum dose 15 grammes (3 teaspoonfuls). In order to avoid a deleterious action on the stomach, doses of 10 grammes must be divided into two parts, and those of 15 grammes into three doses, and each are to be dissolved in ¼ litre of water. The artificial salts is to be administered in exactly the same way.

As a menstruum for solution of the Sprudel salts or the artificial mixture either good drinking water can be used, or, very appropriately, Carlsbad mineral water itself, or finally a water acidulated with carbonic acid (Biliner Sauerbrunnen, Vichy, Giesshübler, Krondorfer, Selters, Fachinger, Eau de Vals, Apollinaris, [Londonderry Lithia], etc.). A lukewarm solution should have the temperature of about 30° C., and a warm one of about 50 to 55° C. The warm solution has a less active effect on the bowel movements than the cold. The warm solution should be prepared by dissolving the salt directly in warm water. (By warming the original solution, the bicarbonate is converted into monocarbonate.) Greater intervals between the individual doses are much to the purpose; thus with two doses of 5 grammes each an interval of from 30 to 45 minutes should intervene, and with 15 grammes the first interval should be 30 minutes, and the second, 45 minutes. Not earlier than 60 minutes after the ingestion of the last dose, the first daily meal may be taken.

Action of the Alkalies on the Healthy and the Sick Stomach.

How do the alkalies act on the stomach? Of late years considerably extensive material as to this question has been brought forward, but on account of dissimilarity of the questions under investigation and the procedures in experimenting the results are to be accepted only with great caution. Naturally the physician has an interest in the following questions: Is the action of the alkalies transitory or permanent; has it harmful effects as well as beneficial ones; may we prescribe them with impunity over a long period of time, and in what doses? For the solution of these problems, investigations were naturally first carried out on the healthy. Such experiments have been carried on by du Mesnil, Linossier and Lemoine, Schüle, Reichmann, Hotellier and others. Apart from slight variations they proved that small doses increase the secretion of hydrochloric acid, larger doses restrict it, but finally furnish an incentive to renewed acid secretion. These experiments, however, do not follow the same course as in the sick. A patient takes the alkalies, a longer or shorter time after eating, sometimes to act as a preventative, but in most cases to cause the temporary neutralisation of an abnormal formation of acid. In order to study the effects of alkalies sick persons are more suitable subjects than the well, and, besides, the medicine must be administered some time after the ingestion of food. With this point in view the investigations of Reichmann and Linossier[18] were instituted, and at the present time they give us probably by far the best idea of the nature of the action of the alkalies upon the diseased stomach.

Reichmann first determined that alkalies given when fasting do not call forth an increase of the gastric juice. Then, again, no special influence was shown when they were taken before meals. On the other hand, the action of 2 to 4 grammes of sodium bicarbonate administered one-half hour after a test-breakfast was very evident: the free hydrochloric acid was considerably diminished or had disappeared altogether. Finally, Reichmann studied the action of the long-continued use of sodium (4 to 8 weeks) and found absolutely no alteration of the secretion of gastric juice.

These experiments of Reichmann's stand harshly in opposition to those of Linossier.[19] This author, in very circumstantial experiments, found that in addition to the direct (stimulating) action of the bicarbonate there is also an indirect action, namely, that this indirect action is first a stimulating one, for the total acidity and the free hydrochloric acid are increased. By further continued use, however, the respective values fall, and that, in the opinion of Linossier,[20] through accumulation of alkali in the blood.

Very similar are also the experiences of Hayem,[21] who found in cases of hyperchlorhydria after treatment with small doses of alkalies (4 to 8 grammes one hour before the meal), an increase of the secretion of hydrochloric acid, and after large doses (10 to 16 grammes during the meal), a decrease. Accordingly, these results agree to a certain extent with those obtained with the use of Carlsbad water or salt solutions.

How does this agree with the observations in daily practice? The only sure fact is the generally known experience that the alkalies, taken in sufficient quantity, neutralise wholly or partially the secreted hydrochloric acid. The experiences that a patient with considerable superacidity always needs larger doses of the alkalies to satisfy the excessive acids seems to speak further for the assumption of the exciting action. On the other hand, the above-mentioned view of the gradual decrease of acid through the continued administration of the alkalies finds no support i the observation of daily practice.

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[18] Reichmann, Archiv für Verdauungskrankheiten, 1895, Bd. 1, Heft 1.

[19] Linossier, Bullet. de thérap., 1896; referred to in Maly's Jahresb. für Thierchemie, 1898, P. 396.

[20] Linossier, *l.c.*

[21] Hayem, Leçons de thérapeutique, 1893, Series 4.

We may interpose that perhaps the doses which these patients use do not suffice for the complete neutralisation of the acid. But such is not the case, as they come to consult us as soon as the excessive use of alkalies has reached an almost insuperable height.

On the other hand, we cannot speak of a greatly harmful influence of the alkalies; otherwise we would have much more frequently an opportunity to make such observations, considering the widely prevalent misuse of the alkalies.

These experiences, in conjunction with the investigations mentioned above, lead to the result that sodium bicarbonate, and by analogy, doubtless, the other alkalies, act as valuable symptomatic remedies for the removal of excessive production of acid, and that even when they are used for a long time in positive superacidity, they exercise no harmful action, neither do they exhibit a direct and permanent curative effect.

Administration and Dosage of Alkalies.

There are two groups of the alkalies, a practical separation of which is of a certain value: alkalies made with carbonic acid and vegetable acids, and the earthy alkalies.

Of the first are used, as a rule, sodium carbonate and bicarbonate and sodium citrate, and of the latter I use magnesia usta and magnesium ammonio-phosphate.

The course of the decomposition of sodium bicarbonate in the stomach (on account of disagreeable alkaline taste, the monocarbonate is seldom considered) occurs according to the following formula:—

$$NaHCO_3 + HCl = NaCl + H_2O + CO_2.$$

If we calculate the respective molecular weights, it happens that 2.3 parts by weight of sodium bicarbonate combine with 1 part by weight of HCl. The following formula is true of magnesia usta:—

$$MgO + 2HCl = MgCl_2 + H_2O.$$

Here the 0.55 parts by weight of MgO correspond to 1 part by weight of HCl. This is the formula when magnesium ammonio-phosphate (see below) is used:—

$$Mg\,(NH_4)\,PO_4 + 3HCl = MgCl_2 + NH_4Cl + H_3PO_4,$$

in which 1.25 parts by weight of $Mg\,(NH_4)\,PO_4$ correspond to 1 part by weight of HCl.

From this we see that the magnesia usta neutralises the largest amount of HCl; then follows the magnesium ammonio-phosphate; while of the sodium bicarbonate we use four times as much as magnesia usta and almost twice as much as of the magnesium ammonio-phosphate.

Concerning the latter, which, besides magnesia usta, I have been in the habit of prescribing for years, sometimes in combination with the magnesia, I wish to mention that it is extremely stable, almost entirely tasteless and odourless, and is remarkably well borne.

The dose of the said alkalies naturally varies according to the degree of superacidity, and we would do well in individual cases to calculate it according to the amount of acidity. Of course, in doing so, we consider only the acidity already present, and not that which develops afterward; but nevertheless we may obtain a useful standard for the administration of alkalies.

According to my calculations, the dose of sodium bicarbonate in a superacidity which exceeds 2.5 per mille is 8 to 10 grammes, of magnesia usta 2 to 3 grammes, and of magnesia ammonio-phosphate 4 to 6 grammes. These serve for the large meals; for smaller meals half of this amount suffices. With an acidity of over 3 per mille of HCl, we may give as much as 12 grammes of sodium bicarbonate, and 5 to 7.5 grammes of the magnesia salts. In all these cases we assume 400 cubic centimetres of fluid in the gastric contents, and the neutralisation of all this is provided for. As, however, a part of the alkali passes into the intestine, and a part (at least of the sodium bicarbonate) is absorbed, and as, finally, only the immediate supply of HCl is provided for, the above figures are rather too low than too high.

Moreover these figures should not be taken as an absolute fixed quantity, but only as a standard for the doses to be given in treatment. A certain experimentation with doses would not be entirely out of place.

Apart from the factors mentioned above, the fact as to whether we should administer the carbonate or the citrate of the alkalies or the above-mentioned earthy salts is not without significance. With the use of the carbonates there is the danger of inflating the stomach through the development of gas, and when the use is continued daily, possibly several times, the likelihood of producing an artificial over-distension is not entirely to be ruled out. On this account the vegetable salts of the alkalies and the earthy salts are to be preferred to the carbonates when atony of the stomach is perhaps already present. They are also to be preferred in cases of great accumulation of gas in the intestinal tract with a tendency towards constipation; while in the remaining cases we may choose between either.

The Carlsbad thermal salts, and likewise the artificial Carlsbad salts, according to Jaworski, are well adapted especially for the treatment of supersecretion (gastro-chylorrhœa), for insufficiency of the acidity of the stomach and for gastric ulcer (small doses in latter cases), and also in habitual constipation and mucous catarrh.

In dilatation of the stomach, Jaworski recommends the thermal salts only for lavage.

L. Wolff[22] recommends for the treatment of superacidity a salt with a composition similar to the Carlsbad salt. It has the following composition: —

Sodium sulphate	30 gm.
Potassium sulphate	5 gm.
Sodium chloride	30 gm.
Sodium carbonate	25 gm.
Sodium biboracicum	10 gm.*

Wolff gives half a teaspoonful of this mixture three times a day, dissolved in half a glass of lukewarm water and taken fasting, then two hours before dinner and two hours before supper. Through the use of this powder the quantity of gastric juice secreted on fasting diminishes, and likewise the amount of hydrochloric acid of the gastric contents removed at the height of digestion. Formerly I used this mixture of Wolff's frequently with good results, but for several years I have been giving the following mixture for the diminution of an abnormally increased secretion:—

℞ Magnesiæ ustæ,		
Natrii citrici,	aa	15.0
Extracti belladonnæ		0.1-0.2

D. S. One teaspoonful three times a day or one or two hours after each meal.

Under no circumstances should the physician limit himself to the ordering of antacid remedies in cases of excess of acid. The principal factor is a regulation of the diet and of the manner of living. I do not doubt that we can succeed in overcoming the complaints through the latter means alone. Especially is this true of that form of superacidity which is the result of too long a retention of the food in the stomach (motor insufficiency, supersecretion, benign stenosis of the pylorus). And also in simple nervous acidity we can attain noteworthy and lasting results with a rational diet.

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* The rather considerable addition of borax, in view of the recent experiences as to the toxicity of borax and boracic acid, had better be omitted in this formula.

[22] Wolff, Zeitschrift für klin. Medicin, 1889, Bd. 16, P. 263.

CHAPTER XIV.

Artificial Ferments.

(Ptyalin, Diastase, Pepsine, Pancreatin, Papayotin, Papain, [Caroid].)

The artificial ferments find their place in therapy where a substitute for defective production of the natural secretions appears desirable or necessary.

Ptyalin, Malt Diastase, Takadiastase.

Ptyalin.—Beginning with the saliva, we saw above (page 129) that in rare cases the salivary diastase is not developed in sufficient quantity. In other cases the excess of acid is so great that the ptaylin is actually destroyed. It is true that with the disappearance of the acid the ferment becomes again active; but nevertheless the activity ceases or is materially reduced under the long-continued influence of the acid. As in these cases the pancreatic diastase also is reduced in its efficiency, at least transitorily, there are indications for the administration of artificial ptyalin or malt diastase. Under all circumstances we do well to add to the ptyalin a little sodium bicarbonate, or, better, sodium carbonate, in order to overcome the excess of acid.

In former years I prescribed for this purpose, in addition to alkalies, also pytalin (0.5 to 1.0 gramme), to be taken half an hour after eating, and I could convince myself by exact quantitative estimations of the amount of sugar as to the activity of the same. Subjectively, too, the administration of diastase is followed by favourable results to the extent that amylaceous food can be taken in far greater quantities than without it.

It is immaterial whether we choose the ptyalin or the malt diastase, except that as the price of the latter is higher and its activity not greater, I usually give the former the preference.

Of late years, I do not prescribe ptyalin. Recent experiences have led me to regard it as totally unnecessary.

Under the name "Takadiastase"* Parke, Davis & Co., of Detroit, have put on the market a new substance for converting starches into sugar. This is obtained from aspergillus oryzæ, one of the fungi used in the rice-wine production of Japan. It consists of a yellowish-white entirely soluble powder, and has the advantage over the other kinds of diastase that it retains its activity in materially higher amounts of acids. While ptyalin and the malt diastase lose their property of converting starches in the presence of 0.01 per cent. of HCl (according to Leo), takadiastase will convert about 60 per cent. of the amylum into maltose even in the presence of 0.05 per cent. of HCl. Consequently Leo[1] recommends takadiastase in cases of hyperchlorhydria, and can thereby obtain an objective improvement in the digestion of starches, as well as (though not always) a diminution of the subjective complaints. Very frequently there is also an improvement in bowel movements. There are also other reports (particularly from American and English authors (J. Friedenwald,[2] Fite,[3] Taylor,[4] Elis[5]), of favourable results from the use of takadiastase where there is occasion to hasten or to better the digestion of starches.

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* **[Takadiastase was first made by Dr. Takamini.]**

Strauss and Stargard[6] had less favourable experiences. They found that the resistance of takadiastase against hydrochloric acid is only slightly greater than that of the saliva. According to this, the administration of takadiastase may be recommended perhaps at the beginning of digestion, and then only when no disturbance of motility with excess of acid exists.

My own experience is limited to one case, that of a woman who used takadiastase as a means against abnormal formation of gas, with alleged great results. There was no superacidity; on the contrary, the secretion of hydrochloric acid was much below normal.

Pepsine.

Since it became possible to isolate the digestive ferment from the stomach of the hog (Corvisart), *pepsine* plays an important rôle in the therapy of gastric diseases.

It is administered in the most various forms and combinations, sometimes as powder and sometimes as pepsine wine; sometimes in the form of pills, some with hydrochloric acid and some without it; sometimes in combination with stomachics, for example, rhubarb, etc., and finally, in combination with other digestive ferments, for example, pancreatin.

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[1] Leo, Therapeutische Monatshefte, December, 1896.

[2] J. Friedenwald, New York Medical Journal, May, 1897.

[3] Fite, Medical News, February, 1897.

[4] Taylor, Lancet, 7 August, 1897.

[5] Elis, Americ. Pract. and News, 1 February, 1898; quoted in Schmidt's Jahrbuch, 1899, Bd. 261, S. 149.

[6] Strauss und Stargard, Therapeutische Monatshefte, February, 1898.

Since pepsine belongs to the official preparations in Germany, we have a uniform agent, the activity of which is warranted to a certain degree. This was formerly not the case, and, as far as I know, does not hold good of some non-German preparations. Certain kinds of pepsine considerably surpass the requirements of the German pharmacopœia, for they dissolve albumen to a greater degree than is required.* For example, the pepsine of Byk is of 150 to 160 per cent. strength, the pepsinum concentratum of Jensen and Langebeck-Jensen is even of 600 per cent. strength.

In general, therefore, we are provided with good and reliable preparations. Less useful are the above-mentioned combinations. Pepsine-wine is very much less active than the pure powder, because only a small quantity of the ferment is soluble even in diluted alcohol. A large quantity of pepsine-wine restricts entirely, according to Werther,[7] the albumen-digesting powers of the normal human gastric juice. According to Hugonneng,[8] this is true of all wines containing pepsine.

Entirely absurd are the combinations with rhubarb, sodium bicarbonate, pancreatin, tinctures, infusions, and similar preparations. The best adapted is the unqualified administration of pepsine in powder form, and I prefer the so-called pepsinum absolutum, which is entirely free from sugar and starch, to the preparations mixed with milk-sugar or starch. The higher price of absolute pepsine is sufficiently offset by the smaller dose necessary. The necessary dose of the absolute pepsine is 0.5 to 1.0 gramme, that of the official 2.0 to 3.0 grammes.

There exist, both within and outside of Germany, a great number of digestive agents, which claim the miraculous. Georges[9] has carefully examined several of these preparations and comes to the following conclusion: "L'action digestive des preparations citées plus haut est nulle dans tous les cas." According to my experiences, this is also entirely true of a great number of these digestive preparations in Germany [and America].

Indications for the Use of Pepsine.

The indications for the administration of pepsine preparations seem clear theoretically: we should order it in those conditions in which the gastric mucous membrane secretes little peptic ferment, or none at all.

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* The second edition of the German Pharmac. and the seventh of the Austrian provides that 0.1 gramme of pepsine with 150 grammes of water and 2.5 grammes of hydrochloric acid of specific gravity 1.124 has to reduce in four to six hours at 40° 10 grammes of boiled hen's egg albumen in pieces as large as lentils to a weak opalescent solution. This pepsine is regarded as of 100 per cent. value.

[7] Werther, Berliner klin. Wochenschrift, 1892, No. 27.

[8] Hugonneng, Lyon médic., 1892, No. 9.

[9] Georges, Archiv de médecine expérimentale et d'anatomie pathologique, 1890, P. 88.

In practice this principle suffers material limitations. We must here distinguish two possibilities. The first consists in the possibility that pepsinogen is secreted exclusively or almost exclusively, and that it only needs the necessary quantity of hydrochloric acid; and the second is that the production of enzymes is lost either to its greater extent or entirely.

In the first case, the prescribing of pepsine is superfluous, because the administration of hydrochloric acid suffices fully to bring about a sufficient production of ferment. Although in the second case the introduction of a peptic ferment into the stomach is very desirable, pepsine is not well adapted, because so great a quantity of HCl is needed to give activity to the pepsine that it cannot be taken without producing subjective and objective disturbances.

In general, pepsine is prescribed much too often. I cannot help ascribing to suggestive activity the remarkable results of essence of pepsine, lactopeptine, etc. One should confine himself, however, to less costly preparations.

Pancreatin and Pancreon.

Pancreatin (more correctly, pancreas powder) was first brought forward on a large scale and investigated therapeutically by Engesser.[10] It was, however, little used in practice as it was thought that it was destroyed in the stomach. In recent years the establishment of precise indications has furthered a new and justified interest in the pancreas ferment.

Just as with pepsine, there are many kinds of pancreas powder, some of which are useful, and others, on account of the total failure of the action of the tryptic ferment, are absolutely valueless. I have had occasion to test the action of the pancreas preparations of a number of apothecaries and have found almost half of them absolutely incapable of digesting. It is therefore incumbent on us, and I never omit this duty, to test the digestive value of a preparation before prescribing it.

Engesser's preparation, although it is not free from gross impurities of different kinds, is an active, and on the whole, a subjectively well-borne preparation. A preparation of pancreatin (pancreatinum absolutum) put at my disposal by Merck, of Darmstadt, is also quite an active tryptic and amylolytic pancreas powder, pretty free from by-products. Besides these, I have tested for their activity Simon's, Witte's, and Schering's pancreatin. All these agents are useful, but their price is higher than that of pepsine.

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[10] H. Engesser, Deutsches Archiv für klinische Medicin, 1879, Bd. 24, P. 539.

The dose of pancreas powder is 1 to 2 grammes, best given in combination with sodium carbonate. I employ it especially in tablet form and prescribe:—

℞ Pancreatin
Natrii carbon. ....aa 0.5
comprim.
Sig.: Two to four tablets one-fourth hour after meals.

Reichmann[11] recommends, in place of frequently inactive preparations of pancreatin, an alcoholic extract of ox-pancreas,* of which a small wineglassful is to be taken after meals.

Recently a preparation under the name of "pancreon" has come on the market. It possesses the characteristic of not being soluble in the gastric juice, and exhibits its enzyme action only in the intestine. Favourable reports as to the action of this agent in stomach affections with absence of HCl have been made by Gockel,[12] Wegele,[13] and Loeb.[14] Other favourable results from the use of pancreon in intestinal diseases, especially in diseases of the pancreas with fat-stools and loss of nitrogen, have often been reported. (Wegele,[15] Salomon,[16] Lenné.[17]) I have at my disposal a similar case of (syphilitic?) pancreatitis, with glycosuria, fat-stools, presence of abnormally abundant muscle-fibres in the stools, in which pancreon undoubtedly had a favourable influence on the last-named factors. Less favourable have been my experiences with pancreon in chronic diarrhœa as a complication of apepsia gastrica. The dose of pancreon is 0.25 gramme two to three times a day before or during the meal, in the form of (purchasable) tablets or powders.

Indications for the Administration of Pancreatin.

The only indication in stomach diseases is *lack or, still better, total absence of HCl in the stomach-contents.* Those cases are especially well adapted in which the likelihood of secretion of the gastric juice may be excluded or is improbable. Here pancreas powder is the sovereign remedy, and the results following it are very gratifying, as I can testify from a great series of observations. The evacuation of the stools is especially favourably influenced through the better proteolysis and amylolysis. In order to avoid deception, I convinced myself in almost each individual case as to the direct digestive action of the pancreatin.

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* According to Reichmann, we cover an ox-pancreas with half a litre of 12 to 15 per cent. alcohol, let it stand in a cool place for two or three days, and filter. According to my experience, it is extremely difficult to avoid putrefactive processes during this procedure.

[11] Reichmann, Deutsche medicinische Wochenschrift, 1889, No. 7.

[12] Gockel, Centralblatt für Stoffwechsel- und Verdauungskrankheiten, 1900, No. 11.

[13] Wegele, *ibid.*, No. 14; Fortschritte der Medicin, 1902, No. 10.

[14] Loeb, Münchener medicinische Wochenschrift, 1901, No. 31.

[15] Wegele, *l.c.*

[16] Salomon, Berliner klinische Wochenschrift, 1902, No. 3.

[17] Lenné, Deutsche medicinische Wochenschrift, 1902, No. 11.

[We cannot help but mention that the medical profession is greatly indebted to Mr. Fairchild for his ceaseless endeavour in improving and furthering the digestive enzymes. Fairchild's preparations have gained the appreciation of the physicians. His pepsine is second to none. Quite recently a pancreas preparation under the name of "holadin" has been put on the market, and from the few experiments I have made, it deserves a thorough trial.

Holadin* is an extract of the entire pancreas gland, presenting all the constituents both of the digestive and the internal secretion. Holadin, whilst possessing great tryptic activity, is of especial potency in respect to the amylolytic and lipolytic enzymes.

All fluid pancreatic preparations are devoid of lipase (fat-splitting enzyme), a constituent of such importance that it cannot be ignored in many cases in which it is desired to utilise the pancreas gland secretion in medicine.

Holadin is now put up in gelatine capsules, each containing approximately 0.2 gramme. The usual dose is one capsule about three hours after meals, and one at bedtime. In cases of diabetes or in serious disorders of digestion attributed to faulty pancreatic functioning, the dose may be gradually increased to two or three capsules at a time.

Besides holadin there are various other preparations of the same make, as, for instance, diazyme (glycerole).

Diazyme glycerole presents the amylolytic enzyme of the pancreas in association with soluble constituents of the gland secretions, devoid of trypsin and lipase, and affords for internal administration, in an isolated form, all the principles concerned in the conversion and metabolism of carbohydrates. The fact that diazyme has no action on proteids or fat, whilst extremely active in the complete conversion of starch into maltose, affords a test of the isolation of amylopsin. In view of the present knowledge of the influence of the pancreas gland secretion in effecting the digestion and metabolism of the carbohydrates, the importance and significance of this new medicinal resource are apparent. Diazyme exceeds in starch-digesting power other preparations. It supplies in a very agreeable and active form the peculiar ferment of the animal digestive secretion upon which depends the conversion of farinaceous foods into a soluble and assimilable form, and thus affords the best resource to compensate for deficient salivary and pancreatic (starch) digestion. Diazyme glycerole is a stable, prepared from the fresh pancreas gland. It should be diluted with several volumes of cold water before being taken, and followed by a draught of water. One teaspoonful constitutes an average dose.

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* Holadin, by the U. S. P. method of assay, exhibits at least four times the starch-converting power of "pancreatin" U. S. P., 1900.

Diazyme seems to fulfill the promises made, at least as far as only a few observations show. Trypsin also has been offered. I may mention in this connection that I also used another preparation called "tryptogen," which had an undoubted favourable effect in the amylolytic digestion in two cases of diabetes mellitus, in one case to such an extent that after its use for a few weeks, hardly a trace of sugar could be demonstrated in the urine.*

There are, of course, an enormous number of digestive preparations on the market in America. Frequently they are a mixture of all substances that might help in the digestion; it seems, it is supposed, that some kind of "selective" digestion is expected. Such preparations frequently contain pepsine, hydrochloric acid, pancreatin, sodium bicarbonate, diastase, etc., etc. I suppose they have quite a sale, as these preparations are made to relieve the physician of making a diagnosis of the real affection, which perhaps may be a chronic nephritis, or valvular disease of the heart, or tuberculosis of the lungs.

Such mixtures of digestive preparations have, I believe, their greatest value in the possibility of using them as vehicles for necessary drugs, as usually they are quite palatable.]

Papayotin and Papain [and Caroid].

These are obtained from the milky juice of the carica papaya, a tree belonging to the family of the papayaceæ, indigenous in the Moluccas (Spice Islands), and thriving especially in the tropics, particularly in Central and South America. From it Wurtz and Bouchut[18] first prepared papain, and Peckolt prepared papayotin. According to the experiments of Wurtz, papain is said to digest up to 1000 times its weight of fibrine; according to those of Peckolt papayotin has likewise a powerful digestive action. In addition, papain and papayotin are said to be active in neutral as well as in weakly acid and alkaline solutions. Nevertheless, control experiments by various investigators (Rossbach, A. Eulenberg, and others) show very irregular results, and here also, owing to the high price of the remedy, it is advisable to test it beforehand.

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* [Even if these preparations have no direct influence upon the digestion in the stomach, we must not forget that, as Boas expresses himself (see footnote, p. 383), "favourable or unfavourable effects of a remedy may manifest themselves only below the stomach, and certainly do actually assert themselves."]

[18] Wurtz and Bouchut, Compt. rend., 1879, Bd. 89.

Finkler[19] recommends the remedy as an aid to the digestion in the stomach, and prefers it to pepsine. Albrecht[20] reports favourable results from the papayotin preparations in children with disturbances of digestion. Rossbach[21] believes that papayotin should be recommended in the absence of the digestive juices. In spite of these results the preparation was not extensively employed until recently. Sittmann[22] pointed out its use in dyspeptic conditions. He used a preparation made by the firm of Böhringer and Reuss, of Cannstatt, which energetically dissolves egg-albumen in neutral, alkaline, and weakly acid solutions. Sittmann saw favourable results from the use of the remedy in doses of 0.3 to 0.5 gramme in acute and chronic catarrhs of the stomach, in dilatation of the stomach, in carcinoma of the stomach, in dyspepsia after chronic ulcer. These results agree with the recent experiences of Max Pickardt,[23] who found the remedy likewise active in acute and chronic gastritis (with the exception of gastritis acida), in cases of motor insufficiency accompanied by subacidity and inacidity, and foremost in atrophy of the gastric mucous membrane and the accompanying diarrhœa. In hyperchlorhydria, papain is, to be sure, not contra-indicated, but is without effect.

These favourable results have not been corroborated by others. Investigations by Hirsch[24] in my laboratory show that papain peptonises food containing albumen, especially egg-albumen, milk and raw meat, better than the acid-free gastric juice, but that the action is far behind that of pepsine. Grote[25] even believes in warning against the use of papain in superacid and ulcerative conditions of the stomach, while it deserves tentative employment in inacidity and subacidity.

Other similar preparations are papoid, recommended by Chittenden, and caroid. Investigations as to the digestive value of caroid, recommended by Chittenden as very active on albuminous bodies and starches, have shown me that it actually is digestive, but does not materially surpass the action of papain.

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[19] Finkler, The Therap. Gazette, Aug. 15, 1887.

[20] Albrecht, Correspondenzblatt für Schweizer Aerzte, 1880, Bd. 10.

[21] Rossbach, Zeitschrift für klinische Medicin, 1880, Bd. 6.

[22] G. Sittmann, Münchener medicinische Wochenschrift, 1893, No. 29.

[23] Max Pickardt, Therapie der Gegenwart, May, 1900.

[24] Hirsch, Therapeut. Monatshefte, December, 1894, P. 609.

[25] Grote, Deutsche medicinische Wochenschrift, 1896, No. 30.

[As regards to caroid, I made use of it in hundreds of cases with good results. The usual dose is 0.15 to 0.2 gramme (larger doses are hardly necessary), and its price is not too high, especially when we consider the small dose. There is one thing which must not be neglected. Frequently caroid is prescribed as powder in capsules, and I cannot emphasise enough that the frequent high-handed addition of some confection or pill-mass on the part of the apothecary must be condemned. I found the made-up pill-mass containing the caroid in the stool of patients; the only thing dissolved was the gelatine capsule. I frequently prescribe caroid in combination with strychnia, especially in atonic and nervous conditions, in the following formula:—

℞ Caroid	6.0
Strychnia sulphate	0.06

Divide in tales doses no. xxx ad capsulas.
Sig.: One capsule at mealtimes, three times a day.

In my experiments I found that caroid acts in any medium, and that the addition of a few drops of lactic acid helps to accelerate the peptonising effect.*]

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* [The latter experience might be of a certain value in cases of cancer of the stomach, in which lactic acid is present.]

CHAPTER XV.

Bitters and Stomachics.

Bitters.

Certain remedies have had for a very long time the qualities attributed to them of stimulating the functions of the stomach, of furthering the secretion of the digestive juices, and at the same time of increasing the appetite. Just to what extent this is actually true is still an open question. Its solution presents almost insurmountable difficulties. For it is apparent that want of appetite and reappearance of appetite may originate in the most diverse causes; indeed, there is hardly a case in which the action of the stomachic is so clear and indisputable that the results dependent upon the use of it can be traced back.

The experimental investigations have likewise not shown any valuable basis for the assumption of certain local effects upon the gastric mucous membrane, particularly as far as secretion is concerned.

Buchheim and Engel[1] found the bitters indifferent for the peptonising of albumen as well as for the conversion of starches into sugar, and thought to see their material significance in the antifermentative influence that they exert.

Tschelzoff[2] experimented partly on artificial digestive mixtures, partly on animals. The latter especially valuable experiments showed that large doses of the bitter extracts severely inhibit the secretion of the gastric juice, and that medium doses also exhibit a diminution of the secretion. Only small doses bring about transitory increase of the secretion, although without improvement of the digestive power.

Only recently systematic pharmacologic studies of the bitters have been made by Ramm[3] and Bokai.[4] The former, under the guidance of Kobert, found in experiments on animals a striking and very remarkable effect of the bitters upon an increase of the leucocytes and (to a slight extent) also of the erythrocytes. Further, there was noted an increase in the strength of the normal contractions of the stomach and intestines. The investigations of Bokai and his followers showed that the bitters (gentianin, erythrocentaurin, quassin, absynthin, lupulin, cetrarin, columbin, condurangin) slightly hinder the artificial gastric digestion, but they do not influence pancreatic digestion. They do not possess antibacterial virtues, but on their use the conjugate sulphates of the urines are diminished. Some of these remedies (cetrarin, absynthin, columbin) also bring about an energetic action of the intestinal peristalsis, while quassin shows only slight effect.

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[1] Buchheim and Engel, Beiträge zur Arzneimittellehre, 1849.

[2] Tschelzoff, Centralblatt für die Medicin. Wissenschaften, 1886, No. 23.

[3] Ramm, Kobert's historische Studien II P. i., according to Virchow-Hirsch Jahresbericht, 1890, Bd. 1, P. 442.

[4] Bokai, Magyar orvosi Archiv, according to Centralbl. f. klin. Medicin, 1894, No. 11.

The experiments of Jaworski[5] and v. Korczynski[6] on human beings have likewise shown a diminution of the secretion of gastric juice and of peptonisation under the influence of the bitters. From the observation of the last-named author the spices seem to bring about a slight increase of the motility.

For further investigation of this subject we have to thank Stekhoven, and especially Reichmann and L. Wolff. Stekhoven[7] came to the conclusion that none of the bitters used (tincture of quassia, tincture of gentian, infusion of calamus, etc.) produced an increase in the secretion of hydrochloric acid, as long as they do not remain in the stomach for longer than one hour; when infusion of calamus has been there for 1½ hours, a somewhat stronger hydrochloric acid production is called forth.

Reichmann's[8] very thorough and careful investigations with various bitters led him to the conclusion that introduction of the bitters into a fasting, non-digesting stomach brings about a slighter secretion of gastric juice, than the ingestion of distilled water does; in numerous cases gastric juice was not attained after the use of bitters, although it was when distilled water was used. In cases in which distilled water failed to bring forth a secretion of gastric juice, bitter remedies did not succeed either in doing this. After the disappearance of the bitters, as a rule, there is an increase in the glandular activity, and in consequence an increased secretion of gastric juice.

In cases of normal digestive activity, these remedies bring about no alteration of the conditions of the secretions. When, on the other hand, acid gastric juice (containing, however, no HCl) is secreted, and when this gastric juice contains only little, if any, peptone, after the administration of bitters (especially absinthe) a higher degree of acidity with a distinct reaction of HCl and peptones could be demonstrated in the gastric contents. In cases of lost function of the glands, it was not possible, by administration of the bitters, to bring about an active gastric juice containing HCl. *When there is a supersecretion, the bitters increase the acidity still more. Bitter remedies do not produce an alteration of function in either healthy or diseased stomachs.*

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[5] Jaworski, Zeitschrift für Therapie, 1886, No. 23.

[6] v. Korczynski, Wiener medicinische Presse, 1901, No. 11.

[7] Stekhoven, over den infloed van eenige stoffen op the soutzuursechretie Weekbl. v. het. Nederl. Tijdschr. voor Geneesk, 1887; ref. in Schmidt's Jahrbuch, Bd. 219; P. 42.

[8] Reichmann, Zeitschrift für klinische Medicin, 1888, Bd. 14, Heft 1 and 2.

The investigations of Wolff[9] on this point came to approximately the same result, when strychnine, the bark of condurango, and bile were tested. Wolff found that condurango was absolutely without influence on the gastric juice, but that strychnine perceptibly increased the glandular secretion in certain cases.

Penzoldt also found that with rhubarb, condurango, and other drugs a curtailment of the time of gastric digestion is not attainable.[10] Of greatest interest are Pawlow's[11] experiments with amara, which, to be sure, were concerned only with animals, but which furnish an explanation of the contradictory experimental results.

> "According to the universal opinion of the earlier and later physicians, the bitters increase the appetite; and if this be so, everything is said. They are, in consequence, real secretory stimulants, since the appetite is the strongest of all stimuli to the digestive glands. It is, however, not at all strange that this had not previously been observed in the laboratory. The bitter substances were either introduced directly into the stomach of perfectly normal animals, or else injected into the circulation. The action of the bitters, however, is principally associated with their effect upon the gustatory nerves, and it was not, therefore, without some reason that this large group of remedies, consisting of substances of the most varied chemical composition, were grouped together mainly on account of a certain bitter taste common to them all. A person who suffers from a disturbance of digestion has, at the same time, blunted taste, a certain degree of gustatory indifference. . . . . . . It is necessary, therefore, that the gustatory apparatus should receive a strong stimulus in order to restore an active and normal sensation."

Pawlow, therefore, comes to the conclusion that the bitters act not only on the gustatory nerves of the oral cavity, but also on the mucous membrane of the stomach in a special manner, so that sensations arise that carry with them the calling forth of a "passionate longing" for food. Accordingly we have to deal with a psychic effect, which secondarily stimulates the physiologic secretory activity.

Weighty objections, however, have been raised against these views. We see loss of appetite in the most varied stomach diseases, in those with as well as without secretion of hydrochloric acid, and we observe further, an extraordinarily well-preserved appetite where there can be no question of a stimulation of the gastric juice (for example, apepsia gastrica). Why is anorexia present in some cases of carcinoma of the stomach and not in others? Why can the appetite, when missing, never be improved in carcinoma of the stomach? Why have fever patients no appetite, and acquire none in spite of all bitter tonics, while they spontaneously regain the same when the fever ceases? There, even Pawlow's attempt at explanation remains at fault.

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[9] L. Wolff, Zeitschrift für klinische Medicin, 1889, Bd. 16, P. 222.

[10] Refer to Eichenberg, Erlangen Dissertation, cited in Therap. Monatshefte, 1890, P. 60.

[11] Pawlow, Arbeit der Verdauungdrüsen, Wiesbaden, 1898, P. 182 (translated by W. H. Thompson, London-Philadelphia, 1902, P. 138).

According to my convictions, the assumption of an appetite-increasing action of the bitters, I will venture to assert, is by no means assured. For just in such cases where we would need them the most (*i.e.*, in nearly all pronounced cases), according to my experience, they fail almost always. In the milder cases, for instance in acute gastritis, a sort of *self-regulation* enters into the case, which then apparently coincides with the use of the amara.

On the whole, therefore, the use of the bitter tonics in diseases of the stomach is very uncertain and undeterminable. They may act at one time and fail at another. In such cases we must rely merely on trial tests.

Stomachica (or Digestives).

By this term is meant substances which, similarly to the bitters, influence favourably the act of digestion in particular, and the digestive process in general. Of the substances that are declared to possess this action there are, apart from the artificial digestive ferments, innumerable examples on the market, only a few of which, however, will bear a rigorous investigation as to their alleged action.

Here also the scientific foundation for these remedies is weak.

This coincides with the circumstance that opinions as to the capabilities of a stomachic are still extraordinarily divided. A great many authors, especially of recent years, assert that a stomachic, besides increasing the appetite, must also increase the secretory and motor functions of the stomach. Most extreme is Penzoldt,[12] who demands of a genuine stomachic that it must be able to better all the functions of the stomach, including the appetite. I cannot agree to this conception. It might be justified if diminished appetite were always, or even frequently, a sequel or an expression of diminished functional activities of the stomach. This is, however, by no means the case. For anorexia, even in a very high degree, may exist in normal or pathologically irritated digestive functions, in which a stimulation of the latter through stomachics is not sought after at all, and in practice does not lead to the goal. In other cases, for instance, in stenosis of the pylorus with stagnation of the gastric contents, that remedy is to be regarded as a stomachic which reduces or stops the abnormal fermentative conditions, or aids the transition of the gastric contents into the duodenum. In other words, there are, *generally* expressed, really no stomachics in the above sense, but every digestive disturbance requires its own stomachic *adapted to the cause of the disturbances.* In some conditions a stimulant may be indicated, and in others, *vice versa*, a sedative, and in still other cases an antifermentative drug may be the best stomachic. Congruent with this view of the case is the fact, discussed further below in connection with orexin, that certain stomachics act only in certain cases.

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[12] Penzoldt, Therap. Monatshefte, February, 1890.

1. *Common Salt.*—We possess some observations of value as concerns the most commonly used stomachic, namely, *common salt.* It is true the results of these do not agree. The earlier investigators (Lehmann, Frerichs and others) ascribe to the salt a favourable rôle, on the ground of experiments on artificial digestive processes, while Ad. Schmidt, Petit, Wolberg, Pfeiffer, Klikowicz, and Roberts came to the conclusion that the digestion of albumen is impaired by a slight amount of salt (0.1 per cent. to 0.4 per cent.) and is inhibited by a larger amount.

Experiments on the living have verified these facts, and explained them in part; the investigations of Reichmann,[13] especially, showed that after the ingestion of salt acid-reacting gastric contents cannot be obtained, but only a neutral or even an alkaline fluid. Wolff and Schüle,[14] on the ground of experiments on the sick, likewise came to the decision that salt in large amounts decreases the acidity of gastric juice and accordingly the peptonising properties, and under certain circumstances may entirely inhibit the secretion of HCl. According to Schüle, the absorption of sugar is said also to be disturbed.

Contrary to this, the experimental investigations of Cahn[15] and Forster[16] show the great significance of this salt for the secretion of gastric juice. They found that in abstinence from salt the stomach also ceases secreting hydrochloric acid, and instead of the latter no other acid appears at the action of digestive stimuli. With the administration of chlorides in excess the secretion of hydrochloric acid in the stomach immediately begins anew.

My own experiences are concerned with the natural salines (Kissingen Rakoczy, Wiesbaden Kochbrunnen, and Homburg Elisabethbrunnen), and in not too advanced cases of gastritis after a three or four weeks' use of these mineral waters there has been almost without an exception an increase in the amount of HCl. Although I can appreciate the contradictory results of the authors named, I cannot escape from the conviction that small and frequently used doses of chlorides slowly bring about an increase of the glandular secretion. I have had occasion to observe the same effect of Kissingen water drank in Kissingen itself.

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[13] Reichmann, Archiv für exper. Pathologie, 1888, Bd. 24, Heft 1 and 2.

[14] Schüle, Untersuchungen über die Secretion und Motilität des normalen Magens, Habilitationschrift, Berlin, 1895, P. 42.

[15] Cahn, Zeitschrift für physiol. Chemie, 1886, Bd. 10, P. 522-535.

[16] Forster, Zeitschrift für Biologie, 1873, Bd. 9.

In fact, the experimental investigations of Reichmann, Wolff, and Schüle can be reconciled with those obtained by experience. Thus, for example, Schüle (and that contrary to Wolff) found that small doses of salt (5 grammes) would not affect the secretion of HCl. That larger doses bring about a destruction of the HCl secretion is the simple sequel of transudation, which, teleologically considered, is for the purpose of diluting the irritant to overcome irritating action upon the gastric mucous membrane (diluting secretion).

2. *Alcohol.*—As is well known, *alcohol* plays an important rôle as a stomachic, and it is likely that manifold elixirs and tinctures with pompous names owe their principal action to alcohol. Opinions do not as yet sufficiently explain the mode of action of the same. Earlier investigators (Gosse, 1760, Frerichs, 1846) saw in alcohol a means of inciting the secretion of the gastric juice, and Cl. Bernard stated that small amounts of diluted alcohol stimulate secretion throughout the entire digestive tract. On the other hand, however, others (Buchheim, Brinton) ascribed to alcohol a less favourable influence on digestion.

More recent experiments, as far as artificial digestive mixtures are concerned, show that almost without exception there is a perceptible delay on slight additions of alcohol, with larger doses a more or less marked retardation, and with large doses a cessation of digestion.. (Buchner, Schellhaus, Bikfalvi, Ogata, Klikowicz, W. Roberts, and others.)

Less uniform are the results in human beings. Richet found that alcohol increased the acidity of the gastric juice; Kretschy, that even in small quantities it decreased it. Buchner's experiments on the living showed likewise that beer and wine in moderate quantities seemed to retard digestion.

For exhaustive experiments undertaken on the action of alcohol with as much care as criticism, we have to thank Gluzinski[17] and L. Wolff.[18] The former, on the ground of his observations, came to the conclusion that we can distinguish two phases of the digestion with alcohol. During the first, digestion of albumen with normal or even increased secretion of HCl is impaired. After the disappearance of alcohol (second phase) the secretion of HCl rises to twice or thrice the amount found otherwise, and this increase is proportional to the amount of alcohol. The mechanical function of the stomach is restricted only to a moderate degree. After the end of digestion the secretion of gastric juice continues longer than in the absence of alcohol.

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[17] Gluzinski, Deutsches Archiv für klin. Medicin, 1886, Bd. 39, P. 405-430.

[18] L. Wolff, *l.c.*, P. 229.

In consideration of the influence of alcohol on the stomach in pathologic cases, we must distinguish between slight and increased acidity. In the latter case a distinctly pronounced second phase is said to be absent; in other words, there is either a minimal difference in the acidity, or none at all. When the acidity is diminished, alcohol no longer is capable of inciting the secretion, and therefore there is no alteration of the production of acid.

Wolff's experiments culminate in the result that alcohol (that is, cognac in small doses) has a slightly stimulating influence on the acidity (that is, secretion of HCl) in a human stomach, but that larger doses may reduce the acidity and perhaps also the formation of peptones. From these experiments the physiologic explanation of the long-known fact may be given, that after the repeated action of the said article of refreshment (genussmittel) the stimulus of the customary food is not answered with the previous energy on the part of the stomach.

Klemperer,[19] on the basis of his investigations, comes to the conclusion that alcohol does not materially increase the secretion, but that it distinctly stimulates the motor activity.

On the other hand, Wolffhardt,[20] on administration of *absolute alcohol* (15 to 30 grammes) at one time or in small single doses, finds a retardation of the propulsion of chyme, amounting to 30 to 40 minutes longer than normally. On the other hand, experiments with small amounts of cognac (30 to 40 grammes 50 per cent. cognac) taken at one time or in small doses show an acceleration of digestion amounting to 30 to 35 minutes. Red and white wines exert an *accelerating* influence upon the digestion whether they are taken during or before a meal.

These investigations, although they may exhibit many deficiencies in their methods, show at least that *alcohol in moderate quantities has no direct deleterious influence on the digestion in the stomach.* To be sure, it would not matter much whether an increase or decrease of secretion could be proven, for there would be present a better duodenal digestion, which would fully compensate for the deficit in peptones formed.

Incidentally, we consider as significant also the effect of an antizymotic action of alcohol, especially on intestinal digestion, a factor which hitherto is thought of only too infrequently.*

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* The error in the experiments thus far carried out has been that all remedies are regarded only as to their effects on the stomach. We must not forget that the favourable or unfavourable effects of a remedy may manifest themselves below the stomach, and certainly do actually assert themselves.

[19] Klemperer, Zeitschrift für klin. Medicin, Bd. 17, Supplementheft.

[20] R. Wolffhardt, Münchener medicin Wochenschrift, 1890, No. 35.

3. *Creasote.*—Creasote, which has been bequeathed to us from the olden times as an anti-fermentative, is also recommended as a stomachic. G. Klemperer[21] especially ascribed its beneficial effects in phthisis to the favourable influence on the gastro-enteric tract. Of late years, this author[22] has expanded this idea, and has tried to show that the material effect of creasote is to be found in its stimulating effect on the peristalsis; he could also observe a stimulating effect upon the secretion, although this was less apparent. Klemperer used the well-known Bouchard-Fräntzel mixture (creasote, 13.5; tinct. gentian. 20.0; vin xerens. 800.0; alcohol, 200.0).

4. *Orexin* (*Phenyldihydrochinazolin*).—Penzoldt[23] recommended this remedy, a chinolin derivative, as a "genuine stomachic." According to his views expressed above, Penzoldt saw especially favourable results in phthisics and anæmics, in those of poor nutrition, and in after-treatment of convalescents from grave operations who are afflicted with poor appetite.

In patients suffering from true gastric affection, Penzoldt tested the said remedy in only a few cases, on account of its irritating action upon the mucous membrane. Since Penzoldt's recommendations there has been written a whole literature concerning this remedy, the discussion of which would lead too far.

Views as to the value of the agent are divided: The favourable results predominate, however, decidedly, according to Penzoldt, in about half the cases of anorexia. But results are obscured on the positive as well as negative side; as to the former, it is not entirely impossible to exclude suggestion; as to the latter, through the frequently wrong indications. But quite apart from this, there occur, besides the astonishing effects, unexplainable failures.

According to Penzoldt and Kronfeld,[24] orexin causes an increase in the secretion of HCl. It is quite likely that there is also an increase in the motor function. The proper domain of the action of orexin lies in the milder forms of dyspepsia (atony of the stomach, beginning gastritis); in ulcer and hyperchlorhydria it is rather contra-indicated. The remedy has also been frequently used with success in vomiting during pregnancy and in anæmia and chlorosis. The author has given up the orexinum hydrochloricum, formerly recommended by Penzoldt, on account of the irritative symptoms it occasionally was responsible for, and it has been replaced by the orexin base and more recently by the orexin tannate.[25]

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[21] G. Klemperer, Berliner klin. Wochenschrift, 1889, No. 11.

[22] G. Klemperer, *l.c.*

[23] Penzoldt, Therapeutische Monatshefte, February, 1890.

[24] Kronfeld, Wien. klin. Wochenschr., 1891, No. 3 and 4.

The medicine is taken as a fine powder in wafers [capsules], or put directly on the tongue and swallowed with a mouthful of water, and then abundant water, about $^1/_4$ litre, is taken afterwards. The best time for administration is about 10 o'clock in the forenoon. The average dose for adults is 0.3 gramme, which may be increased to 0.5 gramme if the smaller dose does not have the desired effect. If the expected result occurs after a single administration or when it has been given several times, we should dispense with it and see whether the result is lasting. If after five to ten days' employment no effect is seen, we should, after an interval of a week, repeat the trial.

Appendix.

The Significance and Principles of the Surgical Treatment of the Diseases of the Stomach.

The surgical treatment of patients suffering from diseases of the stomach is no longer an exclusive domain of surgery, as in the days when Billroth and Péan first reported the celebrated results of their operations.

Gastro-enteric surgery has risen scientifically and practically to its position as an important branch of the medical art from the framework of merely operative feats of strength, which were only to show what the operative technique could venture to do under the protection of asepsis and antisepsis; and as internal medicine borrows from bacteriology all that which appears to assist in the diagnostics, it must also pay attention to the changes of and progress in abdominal surgery, as far as the treatment and cure of gastro-enteric diseases are thereby actually advanced.

Whether the latter is the case, the views concerning it are as yet discordant. The adversaries of operative surgery point to the general statistics, which still show a great rate of mortality. Nowhere, however, are statistics of less value than in abdominal surgery. What is not here included in the count? Correct and incorrect indications, bad and good technique, lack of asepsis and irreproachable asepsis, suitable and unsuitable after-treatment, results from the early and the present period of abdominal surgery.

It is plain that from such a kaleidoscopic collection the physician can form no correct judgment. This is only possible when the statistics of individual eminent abdominal surgeons are given. If we follow these, we find that there is no doubt that the results in the course of recent years have become considerably more favourable. Very characteristic in this respect are the results of two of our most prominent abdominal surgeons, Czerny and v. Mikulicz. The results in *benign* affections of the stomach in the Heidelberg clinics show in 115 operations 11 cases of death—9.5 per cent. The period 1882-1895 shows in 32 operations a mortality of 22 per cent.; the period 1895-1900, in 83 operations, a mortality of only 4.8 per cent. Peritonitis was twice the cause of death, post-operative hæmorrhage, twice, pneumonia, six times, cachexia and chronic miliary tuberculosis once. At v. Mikulicz's clinic during the period 1890-1897, in operations for 30 benign gastric affections 4 persons died (13.3 per cent.). During the years 1897-1899 there were 40 other operations for benign stomach affections, whereby they had to deal 28 times with open ulcer with or without stenosis, six times with a cicatricial stenosis, four times with obscure cases, and twice with neuroses. Three cases died—7.5 per cent. mortality.

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[25] Penzoldt, Penzoldt-Stintzing's Handbuch der Therapie, 1903, 3 Auflage, Bd. 4, P. 264.

Naturally much more unfavourable are the results in malignant diseases of the stomach. But also in these, as comparative statistics show, there has been in recent years considerable lowering of the rate of mortality. For instance, the mortality in gastro-enterostomy, according to Chlumsky, in the years 1881-1885 was still 75 per cent.; 1886-1890, still 48.4 per cent.; 1890-1896, 36.61 per cent.; and v. Mikulicz's[26] latest results show a mortality of only 28 per cent. What is true of gastro-enterostomy is also true of resection. In the latter cases the loss has fallen from an original 80 per cent. to 25 per cent. (v. Mikulicz). Here we must not overlook the fact that in the cases of gastro-enterostomy we have to deal with the patients in an advanced condition of marasmus, while for resection only cases still in good condition are selected. As v. Mikulicz has convincingly pointed out, these results speak much in favour of resection, as, according to this author, 17 per cent. of the cases were radically cured. A better result could not be reached in cancer of other organs (uterus, rectum, tongue, etc.). All this, however, means only mere figures, which include the purely external result as to life or death, but not as to the definite curative results. In this respect the results in benign as well as in malignant diseases still leave much to be desired, although here also the progress of surgical technique is unmistakable. The advantages and the drawbacks of the operations on the stomach make it incumbent on the physician to take an interest in the present development and building up of abdominal surgery. It is the duty of the internist to bring to the surgeon of his selection the cases suitable for operation; *he has to determine the indication for the operation.* This, however, he is enabled to do only when the diagnosis is made as accurate and sure as possible. If the general practitioner is capable of doing it—and there are numerous physicians who, without being specialists, possess sufficient certainty of diagnosis and discrimination of the indications in even difficult cases—without further circumstances the *rapport* with the surgeon may begin; if he is not capable, a previous consultation with an experienced specialist in gastro-enteric diseases (there is now no lack of them in the larger cities) is very desirable, as Penzoldt also advises. For it is always a sort of "macula levis" for the physician if he prepares the patient for the necessity of an operation or even hints at it, when the surgeon, after a thorough examination, declares the same as non-indicated or not longer indicated.

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[26] v. Mikulicz and Kausch, Handbuch der practischen Chirugie, Separat abdruck.

Only in rare cases the internist will recommend an operation if the condition is not clear. The hope of clearing up the situation by an exploratory laparotomy cannot always be fulfilled. Usually the abdominal incision extends only to such a small area that even during the laparotomy difficulties are encountered. Moreover, too long-continued searching for the diagnosis, the palpation of the various abdominal portions, is not without danger for the patient; at least the narcosis is continued longer than necessary, whereby the total duration and the consequent chances of the operation are influenced. Nevertheless it must be admitted that cases of this kind may also occasionally occur; but as a whole they ought to be limited as much as possible.

Quite different is the matter as to exploratory laparotomy for the sake of determining the method of a procedure to be carried out. In this regard, every operation is at the start an exploratory laparotomy, because, in spite of our diagnostic progress, many conditions may show themselves only at the "autopsy in vivo," which again may have a deciding influence upon the method of operation.

If now we turn to the individual gastric affections in which an operation may, under certain circumstances, be in question, we can here only deal with the discussion of some general points of view; the details are discussed in the second part of this work.

The territory in which abdominal surgery enjoys its greatest triumph is the removal of benign pyloric stenoses, whatever may be their cause. In order to gain a firm foundation for the indication of surgical treatment of such cases, we must ask the question: what does the medical treatment in such cases offer? And here experience teaches us that the surgical treatment of non-malignant pyloric stenosis is indicated only in some of the cases. Not a small number are *permanently* cured through appropriate diet, gastric lavage, and probably also through a compensatory muscular hypertrophy gradually developing under a suitable method of life. As, however, the first-mentioned factors are frequently neglected, or cannot be carried out for a great variety of reasons (social position, etc.), a necessity for an operation appears even in such cases in which a possibility of successful internal treatment exists. Finally, there are patients who will rather run the risk of an operation than to be restricted for many years to a monotonous diet, depriving them from many enjoyments of life.

By no means, however, as many a surgeon at the present time claims, all benign pyloric stenoses must be brought before the surgical forum. At the present state of our knowledge we cannot from the start say of any case whether through appropriate behaviour—in the broadest sense of the word—and rational treatment it may not improve to such an extent that the operation may be omitted for the time being, or at least may be postponed. *The exhaustion of all possible palliative measures is, therefore, for benign gastric affection the most important pre-condition for the indication of a surgical operation.* It is, of course, presupposed that we must not go too far in this respect and allow the patient to come to the highest degree of inanition. [The success of some surgeons, especially also in America, makes it sometimes very difficult for the physician to withstand the pressure for operations; I personally am convinced that many cases of grave appearance may be cured—by "cured" I mean an excellent condition of the patient, and that continuing not only for months, but for years. William L. Rodman, John B. Deaver, and with them many other surgeons, express themselves that in the end the patient, so-called cured, will yet have to come to the operating table; only time will tell; this is sure, that no surgeon will claim that an operation could have been accompanied by greater success than internal treatment had been in curing patients who have gained considerable weight and have remained well for many years, and are still in perfect health after six to eight and more years since their last treatment by the physician.]

As to the kind of operative method, we have to deal principally with two operations, namely, the institution of a gastro-jejunal fistula (gastro-enterostomy) or the establishment of the natural passage through the pylorus (so-called pyloroplasty, after Heinecke-Mikulicz). The forced digital divulsion of the pylorus after Loreta is to be regarded nowadays as out of order, on account of its uncertainty. Which of the above-mentioned methods is to be preferred in the individual case is, of course, a matter to be decided by the surgeon, an individual preference for the one or other method and the results obtained by it probably assisting in making the decision. Still, this much can be said, that gastro-enterostomy is the general procedure which can be carried out in all or almost all cases, whereas the pyloroplasty presupposes a fair mobility of the pylorus and a gastric musculature still capable of its function, factors which are present only in a part of the cases. It is difficult to decide which of the methods is easier performed or requires a shorter time for operation; according to my observations, both methods, a good operative technique presumed, are about equal. In far the most frequent cases of benign pyloric stenoses under my observation gastro-enterostomy was performed.

Besides the cicatricial pyloric stenoses, it is *cancer of the stomach* which most frequently requires surgical intervention. The increasing experiences of the physicians and surgeons have likewise materially elucidated the points in question in these cases. The hope of removing the carcinoma by a radical operation appears at the present time, in the majority of cases, not to be fulfilled. This depends on the following circumstances: first, it is very deplorable that numerous patients seek for medical assistance only at an advanced stage of the disease; then, the diagnosis is made by physicians later than could be possible if all the factors were to be considered. They wait too long for the classic symptoms of cancer of the stomach, and much time has thus been lost. Finally, the disagreement of the physicians as to the usefulness of operation in carcinoma is perplexing to the patient and his friends, and provokes vacillation.

According to my opinion, and even at the present state of our science, the diagnosis "cancer of the stomach" can be made in a great number of cases, if not with certainty, at least with a high degree of probability, at a much earlier stage than is usually done. Certainly, the demonstration of a tumour is no longer any pre-condition for a diagnosis. The total ensemble, particularly and above all the age, the most minute and detailed anamnesis, and the examination of the gastric contents are sometimes much more decisive. The surgical treatment also will be benefited by such a proceeding.

Unfortunately, in the greatest majority of cases of cancer of the stomach, we will have to content ourselves with the removal of the mechanical obstacles through gastro-enterostomy. Although the risks of the operation, to be sure, are greater than in benign processes, and that because we have to deal with underfed individuals, impoverished in blood, and sometimes already in a condition of advanced marasmus, the results, as already mentioned, become more satisfactory under skilful selection of the cases and improving technique of operation. The average duration of life after successful gastro-enterostomy amounts to about seven months. If this period were entirely free of troubles, every case of carcinoma of the stomach with mechanical insufficiency, vomiting and increasing debility, etc., ought to be subjected to gastro-enterostomy; but unfortunately only a number, though it may be considerable, of patients have real benefits, lasting to the end. We should, therefore, have the wisdom to transfer to the surgeon only such cases as possess a certain fund of strength and exhibit pronounced symptoms of gastric insufficiency which can be removed either not at all or but immaterially through palliative treatment.

Other operative methods of the carcinoma, such as curretting, etc., have not been adopted by the surgeons in the treatment of cancer of the stomach.

We must mention the institution of a *gastric fistula* in cardiac cancer (gastrostomy). This is to be considered mostly only when liquid nourishment cannot enter the stomach or only partially so. The chances would be more favourable at an earlier stage, when pulpy substances meet with difficulties on their passage into the stomach. In spite of the technique likewise highly developed in this direction, the benefit of gastrostomy *quoad vitam* is not very great: the life is prolonged for about three to five months only in rare cases. At the same time we must consider that the patients are at least not subjected to the cruel fate of starving to death.

An indication belonging only to recent times is formed by the surgical treatment of gastric ulcers. Eminent clinicians, such as v. Leube,[27] v. Mikulicz,[28] Körte,[29] Krönlein,[30] [Rodman, Mayo, Keen and Tinker], and others, the one from the medical standpoint, the others from the surgical standpoint, have reported their abundant observations on this subject.

These lead to the following results, which in principle are uniform.

The indication for the operation is plain and undisputed when perforative peritonitis develops, provided that the perforation occurs when the stomach is full, and the time after perforation does not materially surpass ten or twelve hours. The chief indication for non-acute cases is presented further by the cicatrised stenosis of the pylorus, whether remaining after the cure of an ulcer or complicated with a non-cured cancer. Another indication is given in incessant, repeatedly-recurring hæmorrhages, which cannot be controlled by palliative treatment, as well as in perigastritis, adhesive exudations around the stomach, and subphrenic abscesses. A relative indication for the operation is also present when an ulcer, in spite of repeated and rationally carried out "cures," reduces the bodily strength of the patient in a threatening manner by provoking always and anew pains and vomiting.

An indication for operative intervention (gastrolysis) is also given in adhesions of the stomach with other organs. Unfortunately the diagnosis of such partial adhesive exudates is rather difficult, and although some symptoms (particularly pain on retraction of the left costal arch) have been mentioned, especially by Landerer, they are not sufficient for a positive diagnosis. On the other hand, the extent and the duration of the complaints may require an intervention, and indeed in several of my observations the patients themselves demanded it. When the arsenal of our dietetic and medicinal treatment has been exhausted, and the patients through their suffering have to withdraw from their occupation and lose weight, I regard an exploratory laparotomy as justified, even at the risk that its result will be negative.

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[27] v. Leube, Mittheilungen aus den Grenzgebieten der Medicin und Chirurgie, 1897, Bd. 2, Heft 1 and 2.

[28] v. Mikulicz, *ibid.*

[29] Körte, Deutsche medicinische Wochenschrift, 1901, No. 12; see also Körte and Herzfeld, Archiv für klin. Chirurgie, Bd. 63, S. 1.

[30] Krönlein, Mittheil. a. d. Grenzgebieten der Medicin u. Chirurgie, 1899, Bd. 4.

Finally, we have to make mention of an operation, the justification of which has still to be proved; namely, *jejunostomy*, introduced by Maydl[31] into surgery. The operation in question may be performed in carcinoma (instead of gastro-enterostomy, Maydl), then as temporary operation in severe corrosion of the œsophagus and stomach, in gastric hæmorrhage not to be controlled in any other manner, very rarely in cicatrised pyloric stenosis when the remaining gastric wall does not permit gastro-enterostomy or pyloroplasty, and finally, in grave cases of gastric ulcer which can be cured only by total elimination of the stomach. (v. Mikulicz and Kausch.[32]) As a whole, the indication is accordingly, apart from the carcinoma, in which the majority of surgeons reject it, a very limited one.

[Still another operation (gastropexy) ought to be mentioned, which of late years has been performed by various surgeons (Duret, Davis, Beyea, Bier, Rovsing, Hartmann, Coffey, Edward Martin, Charles Mayo) to relieve the condition of gastroptosis. According to Beyea's report, the surgical treatment for gastroptosis is indicated in suitable cases, and the indication becomes a necessity for many patients who are compelled to work for their necessities of life.]

The foregoing short review shows the manifold progresses which modern abdominal surgery offers. Internal medicine acknowledges the same gratefully by transferring suitable cases in increasing number to their treatment; but it has also its duty to protect its possession against excessive claims. It will be best able to do so by improving and perfecting its own diagnostic and therapeutic armamentary. *For the highest task of therapeutics does not consist in inventing new operations, but in making them dispensable.*

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[31] Maydl, Wiener medicinische Wochenschrift, 1892, No. 18-20; Mittheilungen aus den Grenzgebieten der Medicin und Chirurgie, 1898, Bd. 3, S. 532.

[32] v. Mikulicz and Kausch, Handbuch der praktischen Chirurgie; Herausgegeben von E. v. Bergmann, P. v. Bruns, J. v. Mikulicz, 2 Auflage, Separatabdruck, S. 43.

DISEASES OF THE STOMACH

PART II.

SPECIAL DIVISION.

SPECIAL DIAGNOSTICS AND THERAPEUTICS.

CHAPTER XVI.

Acute and Chronic Gastritis.

(Acute and Chronic Catarrh of the Stomach.)

I. Gastritis Acuta, Acute Gastric Catarrh.

(Catarrhus Ventriculi Acutus, Acute Gastricism.)

By acute gastritis we mean a suddenly occurring inflammation of the gastric mucosa, with increased production of mucus, whereby the glandular parenchyma may be affected to a greater or lesser extent.

Four varieties of acute gastritis may be distinguished: (*a*) simple gastritis; (*b*) infectious gastritis; (*c*) phlegmonous gastritis; (*d*) toxic gastritis.

(a) Simple Gastritis.

We may designate as simple gastritis that inflammation which is set up by a single ingestion of food of excessive quantity or of unsuitable quality—in other words, chiefly by errors of diet. To these belong also the ingestion of exceedingly cold or hot beverages; usually excessive quantity coincides with unsuitable quality. Though exposure to cold has often been stated as an ætiological factor of acute gastritis, other causes which were not readily discernible may have frequently been the real factor.

The chief subjective symptoms are anorexia, disgust for ingestion of the usual diet, and not seldom a perverse craving for piquant, acid, or salty foods; nausea, painful contraction of the œsophageal muscles, increased salivation, and finally, vomiting; after this, great prostration, headache, apathy, thirst. These symptoms may be accompanied by annoying fulness and distension in the epigastrium, flatulence, even pain, of either burning, boring or of spasmodic and colicky character, which decreases or entirely disappears soon after repeated eructation, especially after vomiting or abundant defæcation.

Of importance is the condition of the tongue, which is covered with a sordid-gray and slimy fur; fœtor *ex ore* is often present. The vomited material contains more or less large quantities of food, imbedded in mucus and of an intensely sour smell, either entirely unaltered or badly digested.

Vomiting may be repeated. The second time it occurs with much more difficulty; less food remnants are present, and the vomit consists of clear viscid mucus, mixed occasionally with blood; if the vomiting continues, the vomit is mixed with bile, readily recognised by the patient through its bitter taste.* This usually means the acme of the emesis.

The vomited material, or the contents removed through the tube, may show an abundance of organic acids, or may be neutral or alkaline, as the acid may be neutralised by the addition of large quantities of mucus or saliva. Even if the reaction is still acid, there is no free hydrochloric acid present, while, on the other hand, there may be an abundance of lactic, butyric, acetic, and other fatty acids. The reaction is usually neutral or alkaline, if the vomit does not contain any food.

Whether, however, the general view of the secretory insufficiency in acute gastritis is correct, does not appear to me by any means as proven. In the course of many years I repeatedly examined the gastric juice of patients suffering from acute and sub-acute gastritis, and frequently found normal or supernormal amount of free hydrochloric acid, though the patients complained of marked dyspeptic symptoms.

A second symptom is the tenderness in the gastric region. This is diffuse and usually not very intense. The epigastrium is frequently more or less distended.

The condition of the bowels varies. They are usually constipated if abundant defecation has not occurred simultaneously with the vomiting.

The urine exhibits the qualities of the febrile urine, high specific gravity, an abundance of urates, and occasionally traces of albumen, and is diminished in amount.

Fever may be present in very irritable or young individuals to a moderate degree (38.2-38.5° C.), or may be entirely absent. Swelling of the spleen is not present.

The course of a simple gastritis is characterised by the quick disappearance of the symptoms and the immediate transition to the convalescent state. There are, however, exceptions where cases were neglected, and which then immediately assume the character of subacute or chronic gastritis.

Even in slight cases, an acute gastritis may leave an abnormal sensitiveness or loss of appetite of longer duration. The prognosis, however, is, at least in the adult, always good.

Diagnosis and Differential Diagnosis.

As a rule, the diagnosis of a simple gastritis is so easy and evident that the possibility of another disease need hardly be discussed; if in exceptional cases high fever is present, one may hesitate between the so-called gastric fever (febris gastrica) or typhoid fever. The absence of a swelling of the spleen, which is discernible at the onset of typhoid fever, would plead against typhoid, just as well as the rapid decrease of the temperature in contrast to the gradual rise and fall of the temperature in typhoid fever. Of greatest practical importance is the use of the regular thermometer in all cases of protracted anorexia, epigastric oppression, general malaise, and diarrhœa. Cases of so-called walking typhoid fever, which have to be charged far less to the patient than to the physician, would then belong to the rarities. Diagnostically important, also, is the herpes labialis, which would speak against typhoid, though herpes occasionally occurs in the latter infectious disease.

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* [The so-called "bilious attacks" are mere attacks of acute gastritis, probable involving the duodenum, usually caused by ice water, ice cream, ice cream soda, ice-cold beer, ice-cold milk or tea, and the "quick lunch," pies and pastries, candy, etc.]

Treatment.

It is, in fact, not easy to battle against the old jog-trot of modern social life, which leads to overloading of the stomach with unsuitable foods and beverages. Perhaps this may succeed best in persons who usually acquire acute gastritis as soon as they have submitted to the seduction of an opulent feast or a drinking bout.

As long as the stomach is full, and nausea, eructation, and retching are present, an emetic or the mechanical removal of the decomposed ingesta is the most important condition for a cure. We may succeed in this frequently by the administration of warm water, to which bicarbonate of sodium may be added, or by tickling the pharynx, or in patients who are less inclined to vomit, by the following emetic:—

℞ Tartar stibiat.	0.05
Rad. ipecac.	1.0

M. f. pulv., d. t. d. no. v.
Sig.: 1 powder every 15 minutes until relieved.

or by a hypodermic injection of apomorphia in doses of 0.005 to 0.01. As a rule, however, emetics may be unnecessary. The most simple and most efficient method is thorough lavage of the stomach. The addition of a teaspoonful of sodium chloride and other saline remedies to the water used toward the end of the lavage may be very helpful. As a rule, one lavage will be sufficient. If lavage had to be interrupted on account of great irritability of the patient, or for other reasons, it may be repeated on the following day.

The second task consists of providing rest to the overtaxed organ. This can best be done by *a complete abstinence from any and all food* for one or two days. The stricter the abstinence, the quicker the result will be. If, on account of great thirst or marked prostration, the ingestion of nourishment by the mouth must be resorted to, the patient must restrain himself to liquids exclusively; suitable for this purpose are cold tea without sugar, red wine or cognac, with natural Selters, and lemonade, or weak dilution of hydrochloric, sulphuric or phosphoric acid. Rectal feeding may be given in exceptional cases, as, for instance, in cases of aged and very weak patients.

The time for feeding by the mouth may be indicated by the return of appetite, the disappearance of fever, good sleep, and repaired general feeling. In the first two or three days only soups of slime of barley, rice, or sago ought to be given, to which, however, the yolk of an egg or some crumbs of stale roll or light bread may be added. If the appetite does not return after three to five days, in spite of the disappearance of the catarrhal symptoms, it may be stimulated by weak solutions of chloride of sodium (1 to $1^1/_2$ per cent.), Tokay or port wine (dry), slightly salted caviar, sardelles, oysters, etc.

Frequently, in my experience, patients take up again their normal food after an abstinence of one or two days, a fact for which they usually atone with a gastritis protracted for weeks. In these cases, an especially strict diet has to be resorted to, which must be confined to fluids until the appearance of a good appetite.

The pressure in the epigastrium, if it does not disappear spontaneously, is best cured by hydropathic packs (Umschlag) changed every two hours. In individual cases a continuation of this treatment for several days has been considered necessary.

The stools, as mentioned before, may be copious, or the bowels may be constipated. A special treatment against the former anomaly is usually not necessary, as the diarrhœic evacuations cease spontaneously if strict abstinence is adhered to. But even where copious and frequent defæcation occurs, polypharmacy is not indicated; the opium preparations, especially, are usually superfluous. Constipation, however, may occasionally demand treatment. As a rule, we may get the desired effect by an enema with suitable additions (soap, oil, cod-liver oil, carbonate of soda, glycerine, honey, vinegar); but if not efficient, and if fæcal impaction has persisted for several days, one should not delay by using dubious and slowly acting remedies, but should administer the surely and quickly acting castor-oil or even calomel (0.25 pro dosi, cave stomatitis!). If a copious evacuation of the bowels ensues, the patient may safely wait for three or four days; if then evacuation does not follow spontaneously, an enema is almost always efficient. *One should beware of irritating the weakened alimentary canal at this stage with purgatives and drastic remedies.*

The more we can do without medicinal treatment, the better; but where complications arise, medicines may occasionally be used. In cases of more serious disturbances of the appetite, we may give fluid extract of condurango, one teaspoonful in a wineglassful of water, three times daily before meals, or the fluid extract of bela Indica in the same dose, or dilute hydrochloric acid, ten drops in a wineglassful of water three times a day. If severe pain in the gastric region is present, a hypodermic injection of morphine, or of codeine phosphate (0.03) may be indicated. Suppositoria of codeine and belladonna may be recommended, according to the following formula:—

℞ Codeini phosphorici	0.05
Extracti belladonnæ	0.03
Butyr. cacaon.,	q. s.

M. ut fiat suppositorium.
Sig.: One or two suppositories, if necessary.

(b) Gastritis Infectiosa, Infectious Gastritis.

To the gastritis infectiosa belong those forms of acute gastritis which are caused by an invasion of infectious germs or because their toxins are present either in food and beverages or in the drinking water; to them belong the so-called house and mass epidemics, which have come under observation of various writers, as well as the infections after eating spoiled or decomposed foods. The authorities are divided in their view in regard to the first named variety as a separate and distinct form. Lebert[1] regards it as an infection *sui generis;* Oser[2] and Liebermeister,[3] from their own experiences, join him in this view; Ewald,[4] however, rejects the specificity of the process and holds it as an aggravation of the simple acute gastritis. I am of the opinion that there is a form of acute gastritis, or rather gastro-enteritis, well characterised clinically, which differs so much from simple gastritis by the gravity of the symptoms, and particularly the course of the fever, that as a variety of acute gastritis it merits separate consideration. The fact that the source of infection remains hidden in the majority of the cases observed does not change the matter.

The symptoms being very similar to those of simple acute gastritis, as far as the gastro-enteric canal goes, require no further special discussion. The dissimilarity rests in the general symptoms caused by the high fever; great restlessness, severe headache, even delirium, sleeplessness, increased thirst, cholera-like diarrhœa, rapid pulse, etc.

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[1] Lebert, Die Krankheiten des Magens, Tübingen, 1878, S. 29.

[2] Oser, Artikel Magenkrankheiten in Eulenburg's Realencyklopädie, 2 Aufl., Bd. 12, S. 410.

[3] Liebermeister, Die Krankheiten der Unterleibsorgane, Leipzig, 1894, S. 24.

[4] Ewald, Klinik der Verdauungskrankheiten, 1893, 3 Aufl., Bd. 2, S. 27.

The course is more protracted than in cases of simple gastritis, and may last from three to ten days, and then go, usually by crisis, into the convalescent stage; according to Lebert, some cases may have fever for two or three weeks.

Gaffky[5] gives an interesting and instructive report of a severe enteritis in three persons who drank the unboiled milk of a cow affected with hæmorrhagic enteritis. Gaffky regards as the inciting cause of the said virulent enteritis virulent types of the bacterium coli commune. Gaffky[6] also was the first, and after him van Ermengem[7] and Herman,[8] who found a special type of bacterium coli as the infecting germ in cases of botulism.

Perhaps a similar cause was the factor in the epidemic of gastro-enteritis[9] observed in November, 1888, in Christiania, in the course of which about six thousand persons were affected within three weeks.

As Rosenheim[10] observed, acute gastritis may also be caused by bacteria, as, for instance, thrush fungus.

Differential Diagnosis.

As the symptoms of gastricism are prevalent, we need hardly consider differentially any other infectious disease, at least in the adult, than typhoid fever; but all characteristic manifestations of typhoid are missing in cases of infectious gastritis: swelling of the spleen, rose spots, meteorism, ileo-cœcal gurgling, the well-known typhoid stool, [Widal test] and also the equally typical bronchitis. The fever, too, does not show the classical graduated character, but exhibits even in the beginning the type of a *febris continua remittens.*

Treatment.

The treatment is, as in simple gastritis, chiefly expectative. Only when fever and the consecutive symptoms reach an excessive degree, antipyretic remedies, such as antipyrin, phenacetin, and the old stand-by, quinine, may perhaps be resorted to. In the beginning of the affection, especially in cases with marked constipation, the administration of three doses of calomel (0.25) is not only a sure aperient, but also a remedy influencing favourably the course of the disease.

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[5] Gaffky, Deutsche medicinische Wochenschrift, 1892, No. 14.

[6] Gaffky, Arbeiten aus dem kaiserl. Gesundheitsamt, 1886, Bd. 4.

[7] van Ermengem, Bull. acad. méd. de Belgique, 1892, quoted by Herman.

[8] Herman, Arch. de médecine experimentale, 1899, S. 445.

[9] Husemann, Deutsche medicinische Wochenschrift, S. 960.

[10] Rosenheim, Pathologie und Therapie der Krankheiten der Speiseröhre und des Magens, 2 Aufl., Wien u. Leipzig, P. 236.

(c) Gastritis Phlegmonosa.

Phlegmonous gastritis may occur as an independent malady or as a complication with other gastric affections. Both Gläser[11] and Mintz[12] have observed a case of carcinoma of the stomach complicated with diffuse phlegmonous gastritis. I have repeatedly seen the occurrence of pus in the stomach-contents of patients afflicted with cancer of the stomach.* v. Leube mentions a case of simple gastritis with marked secretion of pus; it is true he does not consider this case as belonging to those of phlegmonous gastritis, while Mintz classifies that described by him under this type. Furthermore, phlegmonous gastritis has been observed as a complication of infectious diseases (typhoid, puerperal fever, pyæmia, variola, anthrax, and articular rheumatism). We may, therefore, speak of a protopathic and deuteropathic phlegmonous gastritis.

Two separate forms of this affection may be distinguished: the diffuse, purulent gastritis and the gastric abscess; either one, however, is a rare disease. Mintz[13] could record in 1892 but 43 cases; since that time a few other cases have been reported, which, however, have not elucidated the obscurity which prevails concerning this subject.

Symptoms.—The symptoms show a picture of acute gastritis with excessive fever and prostration. This gastritis may be followed in a short time (two or three days, Maixner) by general peritonitis.

Diagnosis.

The diagnosis of idiopathic phlegmonous gastritis has not, up to this time, been made with any degree of certainty; it is true, the majority of cases have been recorded at a time when methodic examinations of gastric contents had not been made.

Diagnostic characteristics are pain, vomiting, fever, tympany, diarrhœa, and the consecutive general phenomena.

Pain, localised, usually in the epigastrium, is exceedingly intense; in a few cases it is said to be absent.

Vomiting is hardly ever absent, has the characteristics of peritoneal vomiting, *i.e.,* consists of bile and mucus; pus has not been found in the vomit.

Fever of varying intensity (38° to 41.2° C.) has been observed according to L. Ziemann,[14] occasionally exhibits remissions and exacerbations, and resembles, according to its course, pyæmic fever. Meteorism and diarrhœa were present in some of the observed cases; in others, however, more rare, the bowels were exceedingly constipated.

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* See also Chapter on Cancer of the Stomach.

[11] Glaser, Berliner klinische Wochenschrift, 1883, No. 51.

[12] Mintz, Deutsche Archiv für klinische Medicin, 1892, Bd. 49, S. 487.

[13] Mintz, *l.c.*

[14] L. Ziemann, Inaug.-Dissert., Halle, 1904.

Very marked thirst, dryness, thickly coated tongue are present. The pulse is small, soft; cool peripheral parts, hurried respiration. Later on, the picture may be that of general peritonitis. Blood examinations have been made only twice. In one case of Mintz[15] there was poikilocytosis. Lengemann[16] found increase of leucocytes to 30,400.

The course in cases of gastric abscess usually shows slightly marked characteristic symptoms; an acute and a chronic type of the abscess may be distinguished. In the former type are pains over the whole abdomen, with especially marked localisation in the epigastrium, and vomiting of bile-stained fluid. Death ensues usually after five to fifteen days.

In cases of the chronic type, according to Maixner, vomiting, oppressive sensation, emaciation, and high fever are the chief symptoms. The abscess may perforate to the outside or into neighbouring organs; in the former case, recovery may follow (cases of Deininger,[17] Glax,[18] Kirchmann,[19] Beckler,[20] Rubner[21]); however, death is most frequent.

Differential Diagnosis.

Besides the above-mentioned symptoms, the sequelæ of a previous or still existing infectious disease must be taken into consideration. v. Leube[22] reports a case where all symptoms of an acute, purulent gastritis were present, even pus in the vomit, while the post-mortem examination showed a simple gastritis with marked purulent secretion on the surface of the gastric wall. The diagnosis of a gastric abscess is somewhat easier, especially if a tumour develops in the epigastrium and disappears after copious vomiting of pus; but even in such a case it may, as v. Leube correctly states, be an abscess in the neighbourhood of the stomach.

In differential regard, the greatest difficulties may be exhibited by (1) perigastritis, after perforation of an ulcer or carcinoma in a preformed sac, in which cases the history may perhaps yield a hint; (2) abscesses of the liver; (3) acute pancreatitis; (4) poisoning. In cases of abscess of the liver, chills are frequent, but they may be absent; in cases of acute pancreatitis, in distinction from the phlegmonous gastritis, headache, dizziness, and vomiting of bile are prevalent. History, examination of the vomited matter, cavity of the mouth, and, at last, the absence of other characteristic phenomena of poisoning, will prevent its being mistaken for poisoning.

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[15] Mintz, *l.c.*

[16] Lengemann, Mittetil. aus den Grenzgebieten der Mediz. und Chir., 1902, Bd. 9, P. 762.

[17] Deininger, Deutsches Archiv f. klin. Medizin, 1879, Bd. 23, P. 624.

[18] Glax, Berliner klin. Wochenschrift, 1879, No. 88.

[19] Kirchmann, Wiener med. Wochenschrift, 1880.

[20] Beckler, Bayerisches Intelligenzblatt, 1880.

[21] Rubner, Ungarische med. Presse, 1900, P. 732.

[22] v. Leube, Spezielle Diagnose der inneren Krankheiten, 1 Aufl., S. 237.

Treatment.

The treatment of phlegmonous gastritis is that of peritonitis, namely, symptomatic: ice-bag upon the gastric region, ice pills by mouth, opiates by suppositories, morphine or codeine hypodermically, quinine or antipyrin against fever, and stimulants (wine, cognac, coffee, and camphor injection). In recent time surgical intervention was resorted to three times in phlegmonous gastritis, and that twice (Leith,[23] Lennander[24]) with fatal end, and in a case of v. Mikulicz[25] with favourable result. If a pus tumour develops in the gastric region, especially as the prognosis is very unfavourable, surgical interference, after an exploratory puncture has shown pus, is the most suitable proceeding.

(d) Gastritis Toxica.

A toxic gastritis may be caused by concentrated acids or alkalies, by poisoning with metals and metalloids, by alkaloids, alcohol, chloroform, phenol, nitro-benzol, and aniline, arsenic and phosphorus, hydrocyanic acid; furthermore, through sausage and fish ptomaines, etc. Hayem and Lion,[26] not wrongly, count among the cases of toxic gastritis, those caused by abuse of medicines. The said authors mention the following as belonging to these remedies: copaiba, oil of sandalwood, cubebs, mercury, iodine, sodium salicylate, quinine, antipyrin, colchicum, alkalies in large doses, creosote, iodoform, bromides, chloral hydrate, strychnine, arsenic, drastic remedies, etc.

General Symptoms.

The subjective symptoms consist of severe pains in the gastric region and œsophagus, vomit of bloody quality, increased thirst, prostration, fainting; in severe cases death may ensue from collapse.

The symptoms vary considerably, dependent upon the kind and intensity of the poison acting on the stomach; it is not in the sphere of this book to discuss this separately, but we refer to the text-books on toxicology and forensic chemistry.[27] After the acute symptoms have vanished, they may be followed by chronic disturbances (atrophy of the gastric mucosa, stenosis of the pylorus).

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[23] Leith, Edinburgh Hospital Report, Vol. 4, P. 51, 1896.

[24] Lennander, *l.c.*

[25] Lengemann, *l.c.*

[26] Hayem and Lion, Traité de medécine et de thérapeutique de Bronardel et Gilbert, 1897, T. iv.

Treatment.

The task is to neutralise the poison as much as possible. This can be done either by the administration of suitable antidotes (in acids, alkalies, and *vice versa*, by specific antidotes), or through mechanical removal of the poison by means of lavage. The latter procedure is, however, not free from danger in cases of poisoning with acids and alkalies, on account of threatening perforation of the stomach.

As in cases of poisoning with caustic substances, as already mentioned, cicatrical stenosès at the gastric openings, especially on the pylorus, as well as changes in the gastric mucosa, may follow, we have in the treatment to consider these consecutive conditions. (See chapter, "Motor Gastric Insufficiency.")

II. Gastritis Glandularis Chronica (Chronic Catarrh of the Stomach).

(Catarrhus Ventriculi Chronicus).*

Preliminary Remarks.—If we understand by chronic gastric catarrh an affection which is characterised by chronic dyspeptic disturbances of moderate degree, it would be one of the most frequent diseases. If, however, as is necessary in the present state of science, we circumscribe the definition and distinguish it from similar chronic gastric affections, the chronic catarrh of the stomach is, as I, in agreement with the most experienced clinicians, such as v. Leube,[28] Gerhardt,[29] Oser,[30] and Riegel,[31] emphasize, a less frequent disease than was hitherto claimed. It is true, this is valid only for the clinical conception, while the pathologic anatomical definition goes much further, first, because evidently the first stages of chronic gastritis are clinically not very much marked; then, because catarrhs of the stomach, complicating a large number of chronic diseases, occur only toward the end of life, and, therefore, are not recognised diagnostically.

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* Ewald (Eulenburg's Encyklopädische Jahrbücher, 1892, 2 Jahrgang, p. 473), recently began to object to the term "catarrh of the stomach" as not corresponding to the pathologic processes. We retain the nomenclature because it is clinically naturalised, although we fully coincide with Ewald's objections.

[27] Lewin, L., Lehrbuch der Toxikologie, Wien and Leipzig, 1885; Kobert, Lehrbuch der Intoxicationen, Stuttgart, 1893; Ludwig, Medicin chemie, Wien and Leipzig, 1885; Tr. J. Otto, Anleitung zur Ausmittelung der Gifte, 6 Aufl., Braunschweig, 1884.

[28] v. Leube, Ziemssen's Handbuch der Pathologie und Therapie, Bd 7, Heft 2, S. 53.

[29] Gerhardt, Deutsche medicinische Wochenschrift, 1888, S. 349.

[30] Oser, Eulenburg's Realencyklopädie, 2 Aufl., Bd. 12, S. 417.

[31] Riegel, Die Erkrankungen des Magens, Wien, 1897, S. 559.

Formerly the term "chronic gastritis" was frequently considered as synonymous with chronic dyspepsia. Chronic dyspepsia as an idiopathic disease has disappeared from the modern text-book, but is discussed as a complex of symptoms of the most varying gastric affections by some authors, as, for instance, by Liebermeister, Debove, Rémond, and others. According to my opinion, there is clinically no necessity to consider and to describe chronic dyspepsia as a disease *sui generis*, although in practice we occasionally make use of this term for want of a more suitable one.

Two types, a primary and a secondary gastritis, may be distinguished. The former may be caused and sustained through chronic over-indulgence in food of unsuitable quality or excessive quantity, particularly alcohol; then through deficient teeth, abuse of purgatives, tobacco, and many other noxious alimentary influences. Occasionally, especially in women, cases of gastritis have been observed, though we were unable to give any convincing cause for them.

Secondary catarrhs may develop directly from an acute gastritis, or represent complications of other chronic gastric affections (carcinoma, ectasia, atony, ulcer); finally, they may be the result of conditions of stasis, venous congestions, as they may arise from diseases of the heart, lungs, liver, portal vein, and kidneys, etc. To this class the chronic inflammatory processes would belong, such as are observed accompanying anæmia, chlorosis, leukæmia, pernicious anæmia, syphilis, amyloid, carcinoma, Addison's disease, diabetes mellitus, and others. There is no occasion here to discuss the varied conditions arising from these diseases.[32]

The morbid picture in cases of chronic gastritis varies, and is not sharply circumscribed. As many varieties as are shown in the pathologic anatomical condition of the stomach, are shown *intra vitam*. There, of course, arises the thought to find a clinical picture for the anatomical changes creating it, whereby it would become possible not only to discern the disease itself, but even its type or its stage. The first beginnings of such a clinical anatomical investigation of gastritis have already been made; Jaworski and Korczynsky[33] were the first to travel this road; Hayem[34] followed them; myself,[35] and later Cohnheim,[36] have shown the importance of examination of small particles of the mucous membrane for the diagnosis of gastritis. Hayem probably went the farthest by connecting certain secretory disturbances with definite interstitial and glandular changes.

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[32] See also Hans Herz, Die Störungen des Verdauungsapparates als Ursache und Folge anderer Erkrankungen, Berlin, 1898.

[33] Jaworski and Korczynski, Deutsches Archiv für klinische Medicin, 1891, Bd. 47, S. 758. See also Jaworski, Münchener medicinische Wochenschrift, 1887, No. 8.

[34] Hayem and Lion, Traité de médecine et de thérapeutique de Bronardel et Gilbert, 1897, Tome IV. Compare the careful illustrated description of the histologic changes.

Hayem, distinguishing between a parenchymatous and an interstitial gastritis, describes:—

(1) Gastrite parenchymateuse, hyperpeptique chloro-organique, with two subdivisions, d'emblée and tardive. He means by this term anatomically a degeneration of chief cells, hyperplasia of parietal cells and of the glands themselves, characterised clinically by hyperpepsia with hyperchlorhydria, and he calls it "d'emblée," if the excessive acid secretion occurs at the first stage of digestion; "tardive," if it occurs at a later stage.

(2) Gastrite parenchymateuse muqueuse, meaning what we call gastritis mucipara, exhibited clinically by the absence of hydrochloric acid, and also by hypopepsia.

(3) Gastrite parenchymateuse atrophique, meaning the complete atrophy of the glands without interstitial changes, clinically achylia.

(4) In cases of interstitial gastritis, Hayem distinguishes round-cell infiltration and sclerosis, processes which may occur either limited as such, or together with parenchymatous changes. Clinically, they show the picture of hypopepsia in the highest stages of apepsia.

It appears to me doubtful whether this attempt, considering the limited experience, is sufficiently founded at the present time. Besides, the careful histologic researches of Lubarsch[37] prove how hazardous the demonstration of generative or hyperplastic changes in the gastric mucosa may be; for it is, as Lubarsch states, not at all permissible to infer a perfect functional condition from a perfect or almost normal appearance of the particular cells; but on the other hand, we must not conclude that in cases of a normal morphologic appearance of specific elements and functional deficiency, there may not be a considerable disturbance in the biology of the cells. But whatever it may be, the road which these investigations take is the only one which promises light in regard to the various forms of gastritis.

As in cases of catarrh of other glandular organs, one of the best criterions of gastritis is increased secretion of mucus; but there are, without doubt, types of chronic gastric catarrh in which this is absent or is developed but to a slight degree.

Former researches lead almost unanimously to the conclusion that a characteristic sign of chronic gastritis is either the absence or deficiency of hydrochloric acid. This fact, however, is, according to my recent observations, undoubtedly incorrect. *There is clinically a well-characterised form*

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[35] Boas, Diagnostik und Therapie der Magenkrankheiten, I, 4 Aufl., S. 238.

[36] P. Cohnheim, Archiv für Verdauungskrankheiten, 1896, Bd. 1, S. 274.

[37] Lubarsch, bei F. Martius, Achylia gastrica, 1897, S. 155.

*of chronic gastritis, which is accompanied by normal or increased secretion of hydrochloric acid (gastritis acidi or hyperpeptica.)**

According to the clinical phenomena, therefore, we may distinguish the following forms of gastritis: (1) gastritis with superacidity; (2) gastritis with inacidity; and, intermediarily, (3) gastritis with subacidity.

If mucus is particularly prevalent, we speak of gastritis mucosa; if gastritis is far advanced and accompanied by loss of the glandular elements, of atrophia mucosæ or, according to Ewald,[38] of anadenia.

Diagnosis.

The diagnosis can be made from subjective and objective symptoms as well as from its course. The former, generally expressed, are those of "chronic dyspepsia," which term has been used for a long time promiscuously with chronic gastritis. Each of these symptoms requires most precise consideration. We have to consider, in typical forms, disturbances of appetite, sensation of pressure and fulness after eating, eructation, nausea, and vomiting.

The following objective symptoms are valuable for diagnosis: *tenderness on pressure,* distension of the stomach, condition of the tongue, but, above all, the examination of *vomited material or of the gastric contents removed through the tube.*

(a) Subjective Symptoms.

If we base the following description upon a typical case of chronic gastritis with inacidity, the appetite is lessened or varies, "freakish," and directed toward piquant, sour, or salty food; the taste at the same time may be unpleasant, pasty, insipid. The breath is occasionally offensive, caused by decomposition in the epithelium of the mouth.

The irregular appetite can, under no circumstances, be dependent on the anatomical condition of the stomach; otherwise, in all cases of gastritis we would find anorexia. Such, however, is not at all the case. Even in carcinoma of the stomach, in which the gravest alterations of the tissue occur, anorexia is not always observed. Anorexia cannot be dependent, either, on the chemical function of the stomach, as we find severe forms of anorexia in the presence of normal chemical function, while, on the other hand, entirely normal appetite may be met with in the presence of grave destruction of glandular secretion (atrophy of gastric mucosa).

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* Boas, Wiener med. Wochenschrift, 1895, No. 1 and 2. Albu and Koch find fault with the term "gastritis acida," as hereby a pathologic-anatomical term is combined with a clinical term. (Virchow's Archiv, 1899, Bd. 158.) The term is brief and clear, and has also been adopted by others. If any better term should be found, I would be delighted to relinquish the present term.

[38] Ewald, Klinik der Verdauungskrankheiten, 1893, Bd. 2, 3 Aufl., P. 200.

Pawlow's theory also, according to which, as well known, "appetite juice" is produced through the desire for food and the act of mastication, does not hold good, either, for the diseased stomach. Anorexia is certainly not more characteristic of the chronic catarrh of the stomach than it is of any other organic gastric affection. Its chief cause is probably to be looked for in psychical processes, which, as is well known, are of greatest influence upon general sensations.

The appetite may, therefore, be normally good from the beginning—many instances have been observed by me—or it can become so in the course of rational treatment, and remain good for years.

The dyspeptic conditions proper develop following the ingestion of food, and depend, to a certain degree, upon the kind of food. As a rule, they occur after large meals, and to a lesser degree after soups or fluids in general. The *pressure* may occasionally increase to pain, but is always bearable, so that the patients never need interrupt their occupation (important for the differential diagnosis between chronic gastritis, ulcus, and cholelithiasis). In severe cases of gastritis, sensation of pressure may be present, even on an empty stomach.

Only in the severest stages of gastritis, the so-called atrophy of the mucosa (anadenia, phthisis mucosæ ventriculi), paroxysms of *lancinating pains*, usually independent from the ingestion of food, occur.

The *fulness*, too, occurs likewise immediately or only a short time after ingestion, and that even after a quantitatively moderate meal. Occasionally, several hours after the meals active peristaltic unrest, abdominal rolling and rumbling occur, bowels are distended tympanitically, borborygmi follow one another, and then decreasing molestation or one or several thin evacuations.

The *stool* in chronic gastritis, however, is not particularly typical; it may be normal, constipated, or diarrhœic, or may alternate constipation with diarrhœa. Very severe degrees of constipation, as in ectasia or in some cases of ulcus, are not present, according to my experience, but intercurrent diarrhœa is very frequent; there is even a type of chronic gastritis which is constantly accompanied by diarrhœa (Biedert, Appler, Max Einhorn, Wegele, Martius). It is not decided whether we have to deal in such cases with a continuation of the catarrh to the small intestine, or whether the deficiency of the gastric juice and the subsequent entrance of undigested ingesta into the intestine incites an irritable condition. [As far as my experience teaches me, we find, in cases of gastritis, *constipation*, when there is present a state of *hyperchlorhydria*—I do not say "superacidity," because in the latter case—fermentative acidity without HCl or with decreased HCl—there is diarrhœa much more frequent, or almost the rule]. The examination of the fæces exhibits, as observed by Adolph Schmidt[39] and myself,[40] in almost all cases more or less large masses of undigested substances of connective tissue.

Among the classic symptoms of a well-characterised catarrh are *nausea, eructation, retching, salivation,* and *vomiting.* They represent the product of the irritating effect of the undigested gastric contents upon the mucosa. Here, too, the said symptoms occur after a meal which is indigestible for one suffering from catarrh. The disturbances increase till finally, rarely by means of some manipulations or emetics (particularly sodium bicarbonate, Selters, or lukewarm water), the relieving vomiting with immediate improvement follows. In severe cases, the whole graduation of the symptoms may repeat itself during the digestion in the stomach.

The vomited matter, the exact examination of which coincides with that of the gastric contents, is exceedingly characteristic: it consists of *small quantities of a mass, intermixed with much mucus or saliva and undigested or half-digested food remnants, which, being sticky, adheres to the dish,* and can be poured out only with difficulty, and is even more difficult to filter. A short notice is required in regard to the well-known *vomitus matutinus* (water-brash). It is true, this may occur in cases of chronic gastritis and is particularly a frequent accompaniment to the gastritis *ex alcohole;* but this has nothing to do with the stomach proper, it is only a symptom of chronic pharyngitis or even œsophagitis, and ceases after their improvement.

In the text-books, even in the latest ones, we find heartburn described as one of the regular symptoms of chronic gastritis. I paid special attention to this symptom in my large practice, but I encountered it in but rare cases; perhaps more frequently the complaint of heartburn was found in cases of gastritis with superacidity [hyperchlorhydria as well as fermentative acidity].

(b) The Objective Symptoms.

The most important objective symptom is the *development of the disease.* The history shows either the development of the affection as following an acute gastritis, or a slowly progressing course, extending over months or years. Hereby we must take into consideration the already mentioned ætiological factors. Of importance also, is the mild course of the affection (apart from occasional exacerbations) and its relatively slight influence upon the general health, in contradistinction from the malignant gastric affections.

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[39] Ad. Schmidt, Deutsche medicinische Wochenschrift, 1899, No. 49.

[40] Boas, Diagnostik und Therapie der Darmkrankheiten, Leipzig, 1899, S. 228.

In very marked cases of chronic gastritis, the stomach is more or less *tender on pressure;* this tenderness may increase to pain on deep pressure and may thus show a certain slight resemblance to the painful tenderness in cases of gastric ulcer. If, however, we test the epigastric tenderness by means of my algesimeter, we will find that same is considerably less than that of ulcer, that a pressure of more than four or five kilogrammes is borne without annoyance (the algesimetric tenderness in the healthy person amounts to nine or ten kilogrammes). In some cases, the gastric region is distended, especially after meals; in other cases, the intestine appears very much filled with gas.

The appearance of the *tongue* can be turned to account for the diagnosis of chronic gastritis, but to a slighter degree. Though one finds in many cases a grayish-white coating, there are quite a number of patients suffering from gastritis—particularly women—in whom the tongue remains smooth and polished. The description of the coating of the tongue, as given in most of the text-books on pathology, is taken from the gastritis *ex alcohole;* in these cases, pharyngitis and glossitis are almost always present.

The *gastric contents* show various conditions, according to the various stages of the process and according to the particular type of gastritis (gastritis acida, inacida, mucosa, atrophicans). The most important clinical symptom is the finding of *genuine mucus produced by the gastric mucosa.* Already the macroscopically visible *close mixture* of ingesta with mucus arouses suspicion of gastritis, but it alone is not at all decisive as to the origin of the mucus; for in patients with pharyngitis, laryngitis, bronchitis, rhinitis, etc., more or less large amounts of mucus may also be found in the stomach. [Gastritis and enteritis with diarrhœic stools in patients with tuberculosis of the lungs are not infrequent. I do not think, however, that these conditions are tuberculous, but result from large amounts of sputum swallowed, whereby fermentation is set up in the stomach and intestines. If the patient is taught to be very careful, the gastro-enteritis and diarrhœa improve very rapidly.] Of far greater importance is the presence of leucocytes and epithelial cells of the gastric mucosa, which are met with either in form of dense conglomeration or separate and disseminated in the juice of the empty stomach. If either form is found on repeated examinations, the diagnosis of glandular gastritis cannot be doubted; their absence, however, does not exclude the existence of gastritis.

The amount of acidity also varies according to the type or stage of the affection. In cases of *gastritis acida,* we find increased acidity after the test-breakfast simultaneously with increased amount of mucus; even the juice in the empty stomach, in spite of great quantities of mucus, yields the reaction by dye-stuffs and other reagents for hydrochloric acid, more or less markedly positive. As this type of gastritis has been less known up to this time, the following cases may illustrate the clinical picture:—

1. Dr. G., physician, 29 years of age, has been suffering from gastric disorders as a sequel of an acute gastritis lasting a few days. The affection has become gradually worse, and is dependent upon his method of life, in so far as it becomes rapidly worse after any kind of overindulgence in eating, drinking, or smoking. A year ago patient was suffering from malaria. In former years he indulged excessively, occasionally, in wine, and is very much addicted to smoking.

Patient complains of sensation of pressure, fulness, distension after meals, which sometimes increases to most terrible pains. The burning sensation ascending along the œsophagus becomes severe heartburn. The pains are relieved by alkalies, and sometimes by eating. There exists painful belching, and occasional regurgitation of very acid stomach-contents. Very much mucus, no nausea, no vomiting, no gastric pain. Appetite good, bowels regular.

Present state: Patient very well nourished, looks well. Pharyngitis. Edge of the liver below the costal arch, and is distinctly palpable. No other peculiarities. Epigastrium not tender on pressure.

Test-breakfast: Very much mucus mixed with small amount of food-remnants. Congo, tropæolin—positive; acidity, 43.

Fasting stomach: 30 to 40 cubic centimetres of a whitish liquid, mixed with much mucus; marked reaction on congo and tropæolin.

2. William G., merchant, 30 years, of healthy family, took ill from an acute gastric catarrh six years previously. He made a wager that he could drink fifteen glasses of beer within twelve minutes. A short time afterward he took ill from gastric disorders, consisting of anorexia, occasional vomiting, and pressure in the gastric region. Appetite was so lessened that patient could be without food for the whole day. Very rarely bulimia. Frequently vomiting occurred after meals, following the sensation of pressure. The vomited material consisted of moderate quantities of food-remnants mixed with mucus. The sensation of pressure is also present when stomach is empty, increases after ingestion of food, and is relieved by vomiting. Never real gastralgia. Blood has never been observed in the vomit nor in the stool.

Present state shows normal circulatory and respiratory condition. Gastric region everywhere moderately tender on pressure. Algesimeter, 6 kilogrammes. The borders of the stomach normal, after ingestion of fluids, no succusion sound. One hour after test-breakfast, expression. Small amount of irregularly chymified food-remnants extensively mixed with mucus. Filtrate shows pronounced HCl reaction; acidity, 56. The examination of the fasting stomach exhibits about 25 centimetres of glassy, ropy mucus. Reaction markedly acid. Free HCl decidedly demonstrated in the filtrate.

Gastritis acida is frequently met with, according to my observations, in drinkers; it may represent the irritative stage of gastritis, which is followed later by the depressive stage, with loss of hydrochloric acid.

In cases of subacid or inacid gastritis, the amount of free hydrochloric acid is decreased or is entirely absent; but combined hydrochloric acid may be found in larger or smaller quantity. In the same proportion as hydrochloric acid disappears, the peptic power is sinking, too; discs of albumen or particles of meat put into the filtrated gastric contents are digested but slowly or not at all, in spite of addition of hydrochloric acid.

The condition of the ferments stands in a certain, yet by no means constant, parallelism with the amount of hydrochloric acid. In the beginning the rennin is still abundant; but decreases gradually in its intensity, the more the affection is progressing, till finally the rennin-zymogen also disappears. The quantitative examination of rennin-zymogen[41] is, as Bouveret[42] also emphasises, especially on account of its simplicity, a clinically very important criterion of the progress of the affection, and whether a restitution may be expected or not. In the same way, according to Jaworski,[43] the introduction of a weak solution (for instance, one-tenth normal) of hydrochloric acid into the stomach may be utilised, by which a possible pro-enzyme may be converted into an active enzyme. After aspiration of the introduced fluid and after suitably diluting the same, it is readily possible to ascertain the amount of enzymes.

The objective symptoms of the *atrophic gastritis* (anadenia) are, with the exception of the likewise ambiguous lancinating pains, not very marked. The examination of the stomach-contents shows, it is true, deficiency of the secretory function, so-called achylia, in spite of the entirely normal motor function. This, however, as shown by recent investigations, does not at all prove atrophy of the gastric mucous membrane. We can admit such a case only if, in a stomach previously quite intact, a total secretory insufficiency has taken place, through special acute influences, as for instance, poisoning. For further remarks on this subject, I refer to the Chapter on Gastric Neuroses (achylia).

Recently, a characteristic syndrome, consisting of sensation of pain in the abdomen, nausea, and regurgitation of a watery fluid, has been given by Reichmann[44] for the diagnosis of atrophy of the mucous membrane of the stomach (meaning achylia gastrica). This concurrence of symptoms appears at times frequently, at other times less frequently, but usually either before the ingestion of food, or several hours after the meal. According to my observations, Reichmann's syndrome occurs under very various conditions, so that a sure conclusion can hardly be inferred therefrom upon the condition of the secretion of the gastric juice.

The *motor function* of the stomach in cases of gastritis, is usually quite normal, at times even accelerated. Only in a few cases, and after a long duration of the process, we find a decreased motility. But even in such cases, this condition is shown only to a slight degree. Although other authors state it as a common occurrence, I saw but seldom (stenosing gastritis) an ectasia with formation of retention and fermentation resulting from gastritis. Hemmeter,[45] in his excellent work on diseases of the stomach, speaks from the same standpoint.

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[41] Boas, Deutsche medicinische Wochenschrift, 1892, No. 17.

[42] Bouveret, Gazette médicale de Paris, 1893, No. 22.

[43] Jaworski, Verhandlungen des III Kongresses für innere Medicin, 1888. See also Wiener med. Presse, 1888, No. 48 and 49.

[44] Reichmann, Berliner klinische Wochenschrift, 1898, No. 46, S. 1015.

The assertion frequently made by a large number of authors (v. Leube, Oser, Ewald, Rosenheim, and others), that the stomach-contents in such cases are distinguished by an abundance of organic acids, is likewise not conforming to the facts. I believe to have proven beyond doubt that lactic acid is not produced in cases of glandular gastritis.[46] [In cases of this kind, organic acids may be found, though not in abundance.]

Since Trousseau's establishment of the dogma of vertigo e stomacho laeso, "vertige stomacal" is current as an occasional symptom, in our text-books in the chapter on gastritis. Has anybody ever observed it in a case of true chronic gastritis? I had occasion repeatedly to observe cases of vertige stomacal, but without exception only in connection with mechanical gastric and intestinal insufficiency, and Al. Pick[47] agrees with me.

(c) The Course of Chronic Gastritis.

The course of a primary chronic gastritis is characterised by the local disturbances and their effect upon the general condition. The former have been discussed in the section on "diagnosis." The latter are not entirely uniform. In some cases the weight and strength may be affected. In other cases, however, either one may be quite normal and remain so for years; or there may exist (and, as I believe, this is the most frequent case) alternately a good and bad condition.

As a whole, the gastro-enteric canal, and along with it the whole body, stands in a labile equilibrium. In marked cases of long duration, the reaction upon the *psychic sphere* is discernible, although not quite so constant as is usually assumed; there exists inclination to hypochondria, dislike for society and for work, loss of sleep, etc.

In general, however, we may say that chronic gastritis, even in the condition of incurability, shows a benign course as long as not complicated with an intestinal catarrh. The compensation of lost gastric digestion must be a very extensive one, for certainly everybody experienced in the sphere of diseases of the stomach knows many a patient with incurable gastritis, who, in spite of intercurrent harmless disturbances, can stand the usual bill of fare and shows even a considerable increase in weight.

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[45] Hemmeter, Diseases of the Stomach, Philadelphia, 1902, Third Edition, P. 461.

[46] Boas, Zeitschrift für klinische Medicin, 1894, Bd. 25, S. 285.

[47] Al. Pick, Wiener klin. Wochenschrift, 1892, No. 46-47.

A very characteristic instance from my own practice: Stephen F., joiner, 37 years of age, suffering from the time of his youth from gastric disturbances, consisting of regurgitation of food after ingestion; patient reswallows the same, *i.e.*, ruminates. Moreover, frequently, pressure, fulness, inclination to diarrhœa, [*vide* statement, page 408] anorexia. The examination of the stomach-contents (for more than fifty separate examinations) shows absence of free and combined HCl, traces of enzymes, much mucus; even in the fasting stomach an abundance of mucus. Patient is directed to eat slowly and to suppress regurgitation. Patient succeeded in this, and improved considerably since that time, an increase of five kilogrammes in a short time. Patient, under observation for more than three years, *eats the most indigestible foods without any disturbances whatever in spite of an absolute lack of HCl and almost complete absence of enzymes.*

On the other hand, many cases of gastritis, and especially those of the atrophic type, with grave alimentary disturbances, particularly those with pernicious anæmia, have been observed (Austin Flint,[48] Quincke,[49] Nothnagel,[50] Henry and Osler,[51] Kinnikut,[52] Eisenlohr,[53] Ewald,[54] Martius,[55] Schaumann,[56] Max Koch,[57] Knud Faber and C. E. Bloch,[58] Einhorn,[59] and others). The fact that many observations of cases of atrophy of the stomach without nutritive impairment have been recorded, proves that the atrophy alone cannot account for the severe alteration of the blood. On the contrary, an excellent condition of strength and nutrition has frequently been observed. [Three patients with entire loss of hydrochloric acid and enzymes, who have been under observation of D. D. Stewart and myself for about six years, show a very good state of nutrition; one of them (woman, 50 years old) has lately shown signs of tuberculosis.] Indeed, the investigations of metabolic processes made by v. Noorden,[60] Neusser,[61] and Strauss[62] have shown almost perfectly normal assimilation of nutriments even in cases complicated with pernicious anæmia. The finding of the simultaneous atrophy of the small intestine, stated by Nothnagel, Eisenlohr, Martius, and others, has lost considerably in its conclusive force since the more recent investigations of Gerlach, Habel, Heubner, and particularly of Knud Faber and Bloch, who in unison referred the finding of atrophy of the mucous membrane of the small intestine to post-mortem changes.

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[48] Austin Flint, The American Medical Times, 1860; quoted by Hemmeter, Diseases of the Stomach, 1902, Third Edition, P. 849.

[49] Quincke, Volkmann's Sammlung klinischer Vorträge, 1876, No. 100.

[50] Nothnagel, Deutsches Archiv f. klinische Medicin, 1879, Bd. 24, P. 253.

[51] Henry and Osler, American Journal of the Medical Sciences, April, 1887.

[52] Kinnikut, *ibid.*, October, 1887.

[53] Eisenlohr, Deutsche medicinische Wochenschrift, 1892, P. 1105.

[54] Ewald, Berliner klinische Wochenschrift, 1895, No. 45, and 1896, No. 70.

[55] Martius, Achylia gastrica, Leipzig und Wien, 1897.

[56] Schaumann, Zur Kenntniss der sogenannten Botriocephalus-Anämie, Helsingfors, 1894.

[57] Max Koch, Ueber Veränderungen am Magen und Darm bei der perniciösen Anamie, Magdeburg, 1898.

[58] Knud Faber and C. E. Bloch, Zeitschrift für klinische Medicin, 1900, Bd. 40, P: 98; Archiv für Verdauungskrankheiten, 1904, Bd. 10, P. 1; Beiträge zur Pathologie der Verdauungsorgane, Bd. 1, P. 1-83, Berlin, 1906. S. Karger.

[59] Einhorn, Archiv für Verdauungskrankheiten, 1903, Bd. 9, P. 147.

[60] v. Noorden, Zeitschrift für klinische Medicin, 1890, Bd. 17, P. 137.

[61] Neusser, Wiener klinische Wochenschrift, 1899, No. 15.

[62] Strauss, Zeitschrift für klinische Medicin, 1900, Bd. 41, H. 1 to 4, P. 180.

A particular form of gastritis has been described recently by various writers as *gastritis ulcerosa anachlorhydrica* (Nauwerk,[63] Mathieu,[64] Sansoni,[65] and others). This form is distinguished by the fact that larger or smaller gastric hæmorrhages are observed in the course of the disease, without the post-mortem exhibiting any signs of an ulcer. At the present time, it is impossible to tell whether in these cases we have to deal with a complication of gastritis with alterations of the blood-vessels, or with a particular type of hæmorrhagic erosions. The symptomatology of these conditions also is not sufficiently clear.

Diagnosis of the Atypical Form of Chronic Gastritis.

Above we have emphasised the lack of pregnant subjective symptoms in many cases of chronic gastritis. *This fact makes the diagnosis of chronic gastritis one of the most difficult ones of the entire gastric pathology.* The most important symptoms, such as anorexia, pressure, eructation, vomiting of mucus, may be absent under certain conditions; *cases of gastritis may even exist in latent condition for a long time,* till the patients are induced by some incident, such as gross indulgence in eating or drinking, or a stubborn diarrhœa, to have their stomachs examined. On the other hand, it is noteworthy that a chronic gastritis may exhibit the precise picture of nervous dyspepsia (see Differential Diagnosis). The patients complain of bitter taste, occasional pressure or pain, heartburn, have alternately good and bad appetite, may at times readily digest foods which at other times make disturbances, exhibit great weakness, change of mood, insomnia, etc. In other cases patients complain exclusively of intestinal disturbances, flatulency, constipation, inclination to diarrhœa, pressure and tenderness in the intestinal region; in such cases, on exact examination, a marked gastritis is found in addition to enteritis.

For cases of this kind, the examination of the gastric contents is the only way for diagnosis. Without this, it is true, a chronic gastritis may occasionally be guessed, but may never be recognised for a certainty, and an entirely perverse therapeutic road (for instance, to administer alkalies, when hydrochloric acid is indicated) might be taken.

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[63] Nauwerk, Münchener med. Wochenschrift, 1897, No. 35-36.

[64] Mathieu, Maladies de l'estomac et de l'intestin, Paris, 1901.

[65] Sansoni, Archiv f. Verdauungskrankheiten, 1900, Bd. 6, P. 1.

Differential Diagnosis.

Neuroses and carcinoma of the stomach have to be considered particularly in this regard.

(1) *The differential diagnosis between gastric catarrh and gastric neuroses* is rather difficult in atypic cases, sometimes even impossible. Not a single symptom which could not be common to either one! In spite of this there are a number of hints which may serve as a guiding star for diagnosis. First, the course and disturbances. In cases of neurosis, the course is quickly changing, good days alternating with bad ones in a hasty jumble, while in chronic gastritis such marked rapid changes are observed only rarely or not at all. The complaints in chronic gastritis are dependent on the act of digestion, or stand at least with the same in unmistakable connection; this may be the case also in neurosis, but most frequently there is no distinct relation between ingestion of food and complaints. Likewise there is, in cases of gastritis, an unmistakable dependence of the degree of the complaints on the quantity and quality of the food, while such a correlation exists only slightly or not at all in neurosis, though there are also occasional exceptions.

Without doubt, the result of the examination of the gastric contents yields the surest decision, but by no means in all cases. It is decisive as soon as, on examination of the gastric contents from the fasting stomach, or after test-breakfast, *large amounts of mucus, epithelial cells of the gastric mucosa, and leucocytes in large conglomerations are constantly found.* Hyperchlorhydria, hypochlorhydria, and achlorhydria alone may indicate just as well a simple nervous depression (nervous achylia) as a glandular gastritis; the rapid alternate change of hydrochloric acid secretion would rather indicate gastric neurosis. Of great importance, according to my investigations, is the examination of the enzymes. With the exception of the achylia gastrica, discussed in the Chapter on Nervous Affections, absence of enzymes in connection with absence of hydrochloric acid is rather rare in neurosis. In chronic gastritis, too, it is true, enzymes may be found in a normal degree in the beginning of the disease, but as the affection progresses, they gradually disappear, as already mentioned; this gradual disappearance, or more so, the entire absence of enzymes, in connection with demonstrable increase of gastric mucus, indicates, according to my experience, always inflammatory processes in the gastric mucosa. If accidentally, on examination of the gastric contents, a small fragment of gastric mucosa can be obtained, under favourable conditions it may be made evident whether an intact of catarrhal gastric mucosa is present. (Compare General Part, p. 244.)

2. *Differential Diagnosis between Gastritis or Gastric Carcinoma.—*

This differential diagnosis occurs in cases of severe catarrhs of the stomach accompanied by great loss of weight, or of cancer of the stomach without palpable tumour. The subjective symptoms in such cases are of little value, as carcinoma also may be complicated with chronic gastritis. But the gradual course of the chronic gastritis, characterised by slow exacerbations or alternating improvement and deterioration, is of importance if compared to the carcinoma with its onset frequently accompanied by grave general conditions (emaciation, vomiting, etc.) and all this occurring in many cases while the stomach was apparently healthy. The degree of loss of weight and vitality in patients with gastric catarrh is rarely as great as in those with carcinoma. If increase of weight occurs, as it may happen not too seldom also in carcinoma, it does not last long. In these cases, too, the examination of the gastric contents is of greatest importance; in cases of idiopathic chronic gastritis a grave motor disturbance of the function of the stomach either does not occur, or at least very seldom, and the stomach is empty in the early morning, with the exception of mucus or some gastric juice; in cases of gastric carcinoma, a considerable motor weakness develops frequently even in the early stages, and the result is stagnation in the stomach and retention of acid material in the stomach in the morning.

The stomach-contents in chronic gastritis, as a matter of course, only on the presumption of a test-breakfast, are distinguished, as a rule, by the absence of organic acids, particularly lactic acids; cancer of the stomach, however, exhibits frequently even at an early stage, excess of lactic or fatty acids. On the other hand, it is well known that in cases of carcinoma, motor disturbances may be absent for a long time so that—always presumed that no tumour is palpable—the examination of stomach-contents is entirely like that of cases of chronic gastritis. In such cases, the condition of weight in connection with forced nourishment has to be considered. If this increases for weeks accompanied by an increase of bodily strength, it would speak against a malignant neoplasm is spite of the persistence of subjective and objective disorders. Of great differential diagnostic importance, as I could observe, is the examination of the gastric contents and particularly of the fæces for *occult blood*. For, whereas in chronic gastritis, with exceedingly rare exceptions, occult bleeding does not occur, it is most frequent in carcinoma (*vide* Cancer of the Stomach).

Treatment of Chronic Gastritis.

The proceeding must vary according to the affection, whether it is an idiopathic or a secondary process. In the latter case, the removal of the factors which incite the catarrh through passive congestion is the first and most important requirement. In practice, however, there are great difficulties to overcome. Who could cure the secondary catarrh caused through cirrhosis of the liver by removal of the congesting cause; who believes there is good hope for removing the gastritis by administration of digitalis in cases of a severe disturbance in compensation in a mitral insufficiency?

The chances are more favourable in complications with diseases of the *lungs* and stomach. The same medicinal factors which are indicated for the former act also either directly or indirectly upon the digestive tract. Favourable climatic conditions, especially mountain air, a suitable strengthening diet, or, better, a combination of these factors, as they are met with in numerous sanatoria of Germany and Switzerland, have undoubtedly a favourable influence upon the dyspeptic disorders of phthisis.[66] Furthermore, there are mineral waters which exert a healing action upon both lungs and stomach, as, for instance, the alkaline-muriatic springs of Ems, Gleichenberg, Luhatschowitz, Roisdorf, Scawnicza (Magdalen Spring), Selters [St. Louis Spring, Michigan, Saratoga Seltzer Spring, New York], and others. Among the drugs proper, creasote and its derivates (guaiacol, thiocol, creosotal, etc.) have acquired a permanent place as symptomatic remedies in the treatment of phthisis pulmonum complicated with gastric affections.

On an average the treatment will be symptomatic; the remedies to be considered coincide with those administered in primary chronic gastritis.

We have to consider in the treatment of primary chronic gastritis, (1) hygiene, especially hygiene of mouth and stomach, (2) diet, (3) mechanotherapy, (4) hydro-blaneotherapy, (5) physical methods, (6) the medicinal treatment proper.

1. *Hygiene of Mouth and Stomach.*—The majority of people, partly from ignorance, partly from uncleanliness, neglect the hygiene of the mouth. Usually they content themselves with rinsing their mouth once in twenty-four hours. At the same time they are unaware that the chief source of the products of decomposition lies in the tongue; especially the dorsum linguæ is the seat of numerous stagnant products of food and mucus. This may be easily demonstrated to the patient himself, if the fur of the tongue is scraped off by means of a spatula, and the patient is told to smell it. In my opinion, gastritis and dyspepsia are just the reverse of that which was formerly imagined, namely, the sequel of such lingual catarrhs. They may be easily cured by careful cleaning of the tongue with suitable tongue-brushes, so-called, which may be immersed in a 2 to 5 per cent. solution of hydrogen peroxyde. In the beginning such a cleaning of the tongue must be performed three or four times daily; later, twice a day.

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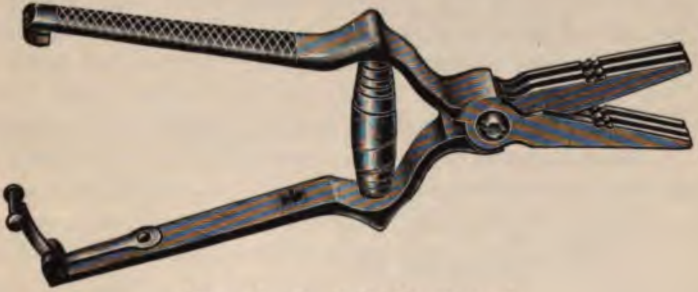
[66] Jaworski, Gazeta lekarska, Warschau, 1892.

2. The *diet* varies according to the type and stage of the disease. Only one function is usually well preserved in all types of gastritis, namely, the *motor function*. The transport of the ingesta, and particularly that of the liquids, is normal or but slightly disturbed. As we know through recent investigations that the disappearance of liquids represents essentially a peristaltic function, nutrition with soups and other liquid foods is decidedly rational in all these cases; but it does not suffice for the necessary economy of the body. It is true, we can increase the food value of the soups by addition of butter, eggs, meat-extracts, and albumoses, but even so very excellent results cannot be obtained for the nutrition. Solid foods must be added. The first condition for their administration is—and this cannot be emphasised enough—a good *set of teeth!* The golden rule of slow eating and thorough mastication must also be impressed upon the patient.

The choice of solid foods and the method of their preparation vary according to the type of the chronic gastritis. If abundant HCl has been found, proteids in shape of eggs, meat, and fish, suitably prepared, are certainly harmless. In cases where HCl secretion is absent, or considerably decreased, the diet is similar, though greater care must be taken in the selection and preparation of meat, and salt and spices in moderate quantities may not only be permitted, but may, on the contrary, be very desirable for the stimulation of the glandular secretion. As the connective tissue is not digested without HCl and also remains undissolved in the intestines, beef may be allowed only after having been rid as much as possible of connective tissue, *i.e.*, scraped meat. Likewise, acids, salads, highly spiced curries, must be avoided. Light vegetables prepared as purées may be recommended. There is no objection to fresh butter. Meat must be prepared in the steam broiler, if possible previously minced or hashed with a so-called "masticator"* [Fig. 55]. A fine division of foods is to be pro-



[Fig. 55.—Carrier's Masticator.]

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* I have frequently made use of "Carrier's Mastikator," for sale at large shops for kitchen utensils [and hospital supplies].

moted by drinking suitable beverages (preferably acidulous waters). *Milk* given methodically in the form of a "cure" is suitable only for the first stages; in advanced cases milk may occasionally be added to soups, cocoa, potatoes, rice, farinaceous dishes, etc., but always only in small amount. Kefyr two days old, or if diarrhœa is present, three days old (½ to 1 litre daily) is preferable. If mucus is very abundant in the stomach, alcohol and tobacco must be forbidden. Fats in a good form, as, for instance, butter, cocoa-nut butter may be advised even in this stage; in order to increase the ingestion of fat in debilitated patients, v. Mering's "Kraftchocolade" (Hauswaldt Vigor Chocolate), which contains 21 per cent. of fat, and the more recent Lipogenchocolate are very suitable.

In the following there is a rational bill of fare for a case of chronic gastritis: —

		Calories.
7 A. M.		
	200 c. cm. milk (135) with 40 Gm. of cocoa (210) plus 30 Gm. of sugar......ca	462.0
Or	50 Gm. of cakes [biscuits]	187.0
	50 Gm. of Zwieback	178.9
10 A. M.		
	50 Gm. of wheat bread with 30 Gm. of butter	343.0
	100 Gm. of calf's brain	140.0
Or	100 Gm. of calf's sweetbread (90).	
	100 Gm. of pike (broiled), (71.75).	
	1 egg (100).	
	50 Gm. of minced ham (225).	
1 P. M.		
	Soup made of 30 Gm. of tapioca, 10 Gm. of butter, and 1 egg	282.0
	100 Gm. of noodles	352.6
Or	100 Gm. of spinach (165), 100 Gm. of bean-purée (193), 100 Gm. of carrots (40).	
	50 Gm. of mashed potatoes (63.7).	
	100 Gm. of meat from breast of spring chicken	106.4
Or	100 Gm. of veal cutlet (230), 100 Gm. of veal (stewed), also squab, game, fish.	
	100 Gm. of farina pudding (omelette, ham and omelette, etc.)	288.0
4 P. M.		
	100 Gm. of milk (with tea), plus 20 Gm. of sugar	147.5
	25 Gm. of cakes [biscuits]	93.5
8 P. M.		
	50 Gm. of wheatbread, plus 30 Gm. of butter	343.0
	50 Gm. of scraped meat	59.5
		2804.5

General directions as to the number of meals and quantity of ingesta cannot be easily given; frequently practice carries our reasoning, though theoretically it may appear well constructed, *ad absurdum*. It may, however, be stated that small and frequently repeated meals are almost always taken care of without difficulty, while copious ingestion of nourishment will incite suffering. In those cases where secretion of gastric juice is absent, repeated drinking of small quantities of acidulous lemonades (hydrochloric and citric acid, etc.) during meals may have favourable action.

Special attention has to be given to constipation, which, however, is never so stubborn as to demand special aperient remedies; in the morning a glass of warm salt-water, salt (as much as on the point of a knife to 250 cubic centimetres water), a small quantity of honey, milk-sugar, mannit or lævulose, plum, fig, or prune preserves, some Graham bread, buttermilk or kefyr (two days old),* and if necessary a glycerine suppositorium or an enema, are usually sufficient.

If inclination to diarrhœa is present, the administration of huckleberry-wine, myrtil confection (one, two, or three times a day), Eichelcacao (acorn-cocoa), or Eichelkaffee (acorn-coffee) is to be recommended; regularly administered soups of rice and barley flour are suitable diets.[67] [Oatmeal soups are less adapted, as they are more conducive to diarrhœa.]

3. *The mechanical treatment* consists of the application of lavage. The same is indicated only when excessive amounts of mucus adhere fast to the stomach-contents. In such cases lavage is most suitably done by warm salt solution (1 per cent.) or with addition of limewater (4 to 5 tablespoonfuls to 1 litre); the capability of the latter of dissolving mucus has been recently emphasised by Harnack.[68] It is best to apply lavage in the morning before breakfast, because the masses of mucus are then easier to remove.

Fenton B. Turck[69] has recommended for the same purpose an apparatus which he called a gyromele (Fig. 56). This consists of a stomach tube which can be rotated by means of a rotary disc and is provided on its end with a sponge. After the easy introduction of the apparatus, the sponge is put in rotation and glides over the mucosa of the greater curvature, the pylorus, and the lesser curvature. The apparatus serves to remove the mucus adhering to the gastric walls, and may also be used for electrisation of the mucosa membrane. The apparatus, the value of which I do not know by my own experience, has not received any special attention up to the present time.

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* [Kefyr (one day old) may be preferable in this condition.]

[67] Boas, Diagnostik und Therapie der Darmkrankheiten, Leipzig, 1901, 2 Aufl., P. 231. Translation by S. Basch, Diseases of the Intestines, New York, 1901, P. 224.

[68] Harnack, Das Kalkwasser, Berliner klin. Wochenschrift, 1888, No. 18.

[69] Turck, New York Medical Journal, February 22, 1896; Wiener med. Wochenschrift, 1895, No. 1 and 2.

According to Aug. Richter,[70] the removal of the gastric mucus is more successful if the tube is introduced only to the cardia, whereby the lavage solution irrigates the gastric walls. Before removal of the water the tube is introduced further, about ten to twelve centimetres, and then the funnel is lowered slowly. [This procedure has always been taught and carried out by the late Dr. D. D. Stewart in the Post-graduate School of the Philadelphia Polyclinic; I may add that in cases where there is a very tenacious mucus, as well as difficulty in removing other gastric contents, the alternate application of warm (even hot) and cold (even ice-cold) water has been of good effect in my practice; it seems that the cold water has a brisk effect upon the gastric walls, whereby mucus is separated from the walls and the other gastric contents are shaken up.]

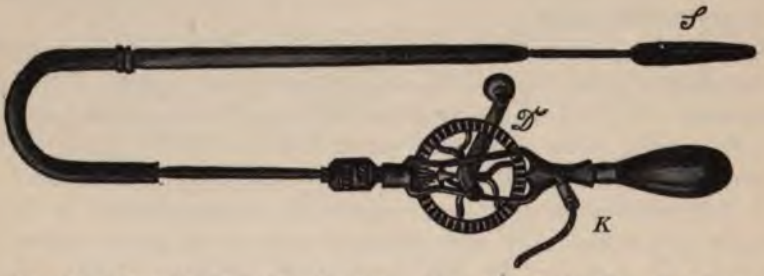

Fig. 56.—Turck's Gyromele. *D*, Rotary disk. *K*, Cord cable. *S*, Sponge.

In other cases, as, for instance, in atrophy of the gastric mucosa or catarrh without formation of mucus, lavage is not particularly indicated.

4. The *balneotherapeutic treatment* is for the purpose of dissolving or mechanically removing the mucus and to vitalise the sunken glandular power. In former as well as in recent times, a great variety of mineral waters has been praised as successful for this purpose. As a whole, it must be said that in spite of all the progress in the chemistry of diseases of the stomach, a real understanding of the action of mineral waters upon chronic gastritis is lacking. The following, however, seems to be certain: small quantities of mineral waters containing sulphate of sodium, and probably also the alkaline and alkaline-muriatic waters, stimulate the glandular function, larger quantities inhibit it; sodium chloride waters stimulate glandular function in all cases where there is still any possibility of function. In the individual cases, for the beginning stages of catarrh as well as for gastritis acida, the sulphate of sodium waters (Carlsbad, Elster,

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[70] Richter, Zeitschrift für diätetische und physikalische Therapie, 1899, Bd. 2, P. 347.

Marienbad, Franzensbad, Rohitch, [Bedford Springs, Pennsylvania]) as well as the alkaline and alkaline-muriatic acidulous waters (Bilin, Vichy, Fachingen, Giesshübel, Krondorf, Vals, Ems, Gleichenberg, Roisdorf, Selters [Londonderry Lithia Springs, New Hampshire]) in medium doses are to be recommended: for the later stages, especially the sodium chloride waters would be suitable. Among the latter, Kissingen (Ragoczy, Pandur), Homburg (Elisabethspring), [Hathorn Springs, Saratoga, N. Y.], have already acquired well-known reputation; Wiesbaden (Kochbrunnen, 60° R.) has begun to join in their success. Furthermore, we have to consider Soden (Milchbrunnen), Mergentheim, Rehme (Bitterbrunnen), Baden-Baden (Hauptstollenquelle), and others. As a matter of economy, the mineral waters may be suitably replaced with the very low-priced Sandow's salts,* which contain the essential ingredients of the respective mineral waters. "Bitter waters," on account of their irritative action upon the gastric mucosa, are best avoided.

It hardly needs special mention that mineral waters must always be administered in combination with the respective diets. The favourable results in Kissingen, Carlsbad, Homburg, Neuenahr, etc., are probably never referred to the use of the mineral spring waters and mineral spring baths by themselves. [The diet is always neglected at American springs; the managers of the hotels usually do not care to have the trouble of a separate diet kitchen; but this negligence is partly or for the greatest part the fault of the physicians, who *do not insist* upon dietetic measures.]

5. *Physical Methods.*—Among them we have to mention, above all, the warm packs (Umschläge), justly liked in the treatment of chronic gastritis. The most suitable time for their application is at bedtime, but they may also, if possible, be applied during the day. Of similar service is also Winternitz's Umschlag (General Part, page 342). These packs are particularly beneficial against the painful sensation of pressure. Other hydrotherapeutic measures, such as half-bath, frictions, Scottish douches, etc., may also be applied to advantage in chronic gastritis, especially in those cases complicated with neurasthenia.

6. *Medicinal Treatment.*—The use of drugs is avoidable in a great number of cases of gastritis, because dietetic and balneotherapeutic measures by themselves are usually satisfactory. It is true, it is not entirely easy to withstand the temptation to use one of the "most approved" of the large number of "stomach remedies." *And yet the cases treated non-medicinally are the most instructive, especially as they also show the patient that his salvation depends essentially on the rest of the diseased organ.*

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* [Similar salts are put on the market by a number of American pharmaceutical houses.]

Occasionally, however, there occur symptoms which require a special medicinal treatment, as, for instance, anorexia, pressure, and intestinal complaints.

Against *anorexia* the above-mentioned sodium chloride waters are very suitable remedies in many cases; where they fail, the other remedies also have only a slightly marked effect. In such cases, I recommend the bark of condurango and columbo (fluid extracts), one teaspoonful before meals several times a day.

Fluid extract of belæ indicæ (one teaspoonful in a wineglassful of water one-half hour before meals, two or three times a day) has occasionally been of really good service to me. In more recent time extractum Chinæ Nanning has been highly recommended by Thomalla[71] as a stomachic, particularly in dyspepsia of chlorotics and alcoholics. This is a fluid preparation with 5 per cent. alkaloid and containing all active ingredients of the red cinchona bark in solution, especially also the cinchona tannic acid, while the inactive substances have been removed. The dose is 15 to 20 drops in water or port wine three times a day.

The importance of creasote as a stomachic has already been mentioned. An addition of tinctura nucis vomicæ may be not amiss; orexine, however, is not indicated in chronic gastritis, on account of the irritative action upon the gastric mucosa. The other bitter remedies, such as tinctura chinæ composita, elixir aurantium compositum, tinctura amara and aromatica, and many similar ones are nothing more than a placebo; a real influence upon the appetite or the inflammatory process is not to be thought of, as I could convince myself in the course of many years.

Since the proceeding of v. Leube,[72] hydrochloric acid has not disappeared from the treatment of the chronic catarrhs against the *painful pressure,* and simultaneously against the flatulency also. I gave my opinion on the HCl treatment in the General Part (page 358). I administer hydrochloric acid in medium doses (10 drops in water three times a day) in initial catarrhs as digestive constantly after meals; in advanced cases, when the peptic enzymes have disappeared entirely or almost entirely, hydrochloric acid has *the function of stimulating the pancreatic secretion* (Pawlow). In these cases the administration of hydrochloric acid on an empty stomach is indicated. Instead of hydrochloric acid, Rob. Flatow[73] recently recommended *acidol* (Betainchlorhydrate) in doses of 0.5 to 1.0 gramme, whereby the smaller dose corresponds to about 5 drops, and the larger dose to about 8 drops of pure hydrochloric acid. The preparation is put up for sale in tablets, is easily soluble in water, and may also be combined with pepsine in a stable form. Flatow frequently used the preparation successfully in nervous subacidity and inacidity. [I have used *chloralbacid* to advantage at various times.] In these cases *pancreas powder, pankreon,* and *papain* (0.3 to 0.5 in tablet form) [as well as *caroid*] are suitable digestive remedies; the latter dissolve most easily meat-albumen, while egg-albumen is dissolved to a lesser extent. The said powders may be administered after meals, either in powder, [in gelatine capsules] or tablet form. (Compare General Part, page 372.)

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[71] R. Thomalla, Therapeutische Monatshefte, 1899.

[72] v. Leube, v. Ziemssen's Handbuch, Bd. 7, Heft 2, P. 75.

[73] Rob. Flatow, Deutsche medicinische Wochenschrift, 1905, No. 44.

Frequently, in cases of chronic gastritis pepsine preparations with or without HCl have been given. The inefficiency of these preparations has already been emphasised.

In most recent time French investigators, relying upon the classic experiments of Pawlow and Schumowa-Simanowska on dogs, were the first to make the trial of administering the gastric juice, thereby obtained, to human beings. (Frémont,[74] Launois, Barth, Legendre, Huchard,[75] A. Robin,[76] and others.) The experiences, known so far, particularly in cases of hypochlorhydria and nervous dyspepsia, seem to be favourable. Paul Meyer,[77] too, it is true on the basis of only one observation, considers the efficiency of the canine gastric juices ("Gastérine Frémont") very hopeful. According to A. Finkelstein,[78] we have in the gastérine a powerful remedy in chronic gastritis; it showed itself very useful, too, in cases of anæmia, typhoid fever (?), even in cancer of the stomach it gave excellent, though but palliative services. According to my opinion, the favourable effect can rest solely in the higher amount of pepsine or in the action of the same, particularly as the pepsine preparations which are in the market prove themselves often entirely valueless. Nevertheless, further trials with "gastérine" ought to be looked for with great interest. [There is no doubt about the greater effect of the natural acid gastric juice if compared with the hydrochloric acid-pepsine mixture.[79]]

Purgatives as well as anti-diarrhœics are, in the treatment of chronic gastritis, not only superfluous (excepting intercurrent instances which occasionally require action) but, in my opinion, directly *injurious.* We stated above for either eventuality the suitable dietetic measures which are sufficient in almost all cases.

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[74] Frémont, Bulletin général de thérapeutique, 1898.

[75] Huchard, Bulletin de la société de thérapeutique, 1899, No. 16 and 17; *ibid.*, 1900, No. 23 and 24, P. 542.

[76] A. Robin, *ibid.*

[77] Paul Meyer, Zeitschrift für diätetische und physikalische Therapie, 1900, P. 220.

[78] A. Finkelstein, Wratsch, 1900, No. 32; quoted in Therapie der Gegenwart, 1900, P. 472.

[79] Albert Bernheim, Pennsylvania Medical Journal, February, 1906.

CHAPTER XVII.

The Round Ulcer of the Stomach.

(Ulcus Ventriculi Rotundum, Perforans, Corrosivum, Pepticum, Rodens).

Preliminary Remarks.—Round ulcer of the stomach is a loss of substance of the mucous membrane of the stomach, with a tendency to progress toward the depth of the mucosa. The losses of substance of the mucosa may be, from the beginning, broad and deep, or may be gradually confluent from several smaller losses of substance. The views on the etiology of the round gastric ulcers vary very widely. It must be assumed as a certainty that the most different causes and conditions may lead to the development of a gastric ulcer. Though it is a fact that gastric ulcers occur in individuals of all ages and all walks of life, it has been shown that certain ages and certain anomalies in nutrition are predisposing factors in the formation of ulcers. In referring, as regards the widely varying theories on the etiological factors of the development of the gastric ulcer, to the large text-books on diseases of the stomach,[1] we will emphasise in the following briefly the most salient points. Above all, it must be stated that there is no uniform ætiology for gastric ulcer which will fit in all cases. If the theories on the ulcer hold such a broad space in our text-books, it is proven that not one of them is satisfactory for the interpretation of the various conditions under which formations of ulcers may develop. The most valid one is still Virchow's[2] opinion that circulatory disturbances in the larger branches of the vessels which supply the gastric walls represent the starting point of the formation of gastric ulcers. But Virchow adds that erosions and bleeding ulcers may develop without venous congestion in acute and chronic catarrhs, particularly in those which are accompanied by severe emetic action and spasmodic constrictions [vomiting during pregnancy].

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[1] Earlier literature, well-arranged, may be found in v. Leube, v. Ziemssen's Handbuch der Speziellen Pathologie und Therapie, Bd. 7, Heft 2. Recent literature is considered by Oser, "Magenkrankheiten," in Eulenburg's Realencyklopädie, 2 Aufl., Bd. 12, P. 462. Compare, besides the text-books on Diseases of the Stomach, the following monographs: Debove & Renault, Ulcère de l'estomac; Bibliothèque médicale, Charcot-Debove, Paris, 1892, (exhaustive, clinical description of our present knowledge of gastric ulcer.) In addition, the works on Diseases of the Stomach by Ewald, Bouveret, Rosenheim, Fleiner, Riegel, Hemmeter, Debove and Rémond, Alb. Mathieu contain the most important literary sources.

[2] Rud. Virchow, Historisches, Kritisches und Positives zur Lehre der Unterleibsaffektionen, Virchow's Archiv, 1856, Bd. 5, S. 281.

Clinic experiences, too, show that the development of gastric ulcers may be based upon chronic inflammatory processes in the gastric mucosa. In cases of this kind, various occasional causes may favour or even incite the development of the ulcer; among them we find mechanical (trauma, foreign bodies), chemical, thermic, bacterial influences, etc.

Moreover, the *secretion of the gastric juice* has certainly to be taken in account ætiologically in the formation of ulcers. While losses of substances in the mucosa heal in a very short time, as Griffini and Vassale[3] have taught by their experiments on animals, the loss of the substance persists in cases of increased acid secretion, or even becomes larger. Whether it is only the *increased* secretion of juice, or whether other certain specific substances, which emigrate from the blood into the glandular system, are to be considered, we do not know.* If once a bleeding has occurred through any of these causes, the circulation in the respective region is easily disturbed. Under such conditions this area stands in the same relation to the gastric juice, particularly if the same is abnormally acid, as albumen introduced in the stomach would stand—it is liable to digestion, and that as far as the circulatory disturbance extends. The formation of loss of substances is started.

As investigations of v. Quincke and Daettwyler,[4] as well as of Mesnil de Rochemont,[5] have demonstrated, certain alterations in the quality of blood have a favourable influence upon the persistence of the existing ulcer.

In reference to the connection of the small hæmorrhagic erosions and the follicular ulcerations with the peptic ulcer, there is diversity of opinion also. In the majority of text-books, the opinion prevails that the latter originate in the small losses of substance. In contradiction, Langerhans[6] recently emphasised that erosions but very exceptionally turn into chronic ulcers. Dietrich Gerhardt,[7] too, has been able to demonstrate only one sure case of transformation of erosion into development of ulcer in spite of several observations of hæmorrhagic erosions.

Clinically, however, it must be stated that hæmorrhagic erosions may exhibit the same symptoms as a peptic ulcer does; in recent times, Hampeln,[8] Pilliet and Deny,[9] A. Fränkel,[10] Dieulafoy,[11] and others have described cases, including autopsies, which demonstrated that fatal hæmorrhages may arise from simple hæmorrhagic erosions just as well as from a developed round ulcer. [Compare William L. Rodman,[12] Gastric Hæmorrhage and Gastric Ulcer; Non-perforating Hæmorrhage.]

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* v. Sohlern (Berl. klin. Wochenschrift, 1889, No. 13) has called attention to the fact that chronic gastric ulcer rarely occurs in Russia and among the inhabitants of the Roehn Mountains, and has attempted to ascribe this rarity to the ingestion of abundant potash of the usually vegetable foods in this region. In my practice, too, the exceedingly rare occurrence of gastric ulcers among my Russian patients has been strikingly observed.

[3] Griffini und Vassale, Beiträge zur pathologischen Anatomie von Ziegler-Nauwerck, 1888, Bd. III, P. 423.

[4] Quincke und Daettwyler, Correspondenzblatt für Schweizer Aerzte, 1875, P. 101.

[5] du Mesnil de Rochemont, Münchener med. Wochenschrift, 1897, No. 51.

[6] R. Langerhans, Virchow's Archiv, 1891, Bd. 124, P. 373.

[7] D. Gerhardt, Virchow's Archiv, 1892, Bd. 127, P. 85.

It is well known that the main contingent of the peptic ulcer is presented by adolescent chlorotic girls. Why is this so? I agree with the view of v. Hösslin,[13] who, on the basis of careful investigation, considers chlorosis as the result of repeated latent hæmorrhages. We know that chlorotic young girls have a particular disposition to hæmorrhages (epistaxis, menorrhagia, etc.). Furthermore, I have often convinced myself that by an exact examination (guaiac test) small quantities of blood have been shown in the vomited material of seemingly normal appearance. Of late, therefore, I consider it of great importance to test the vomit for blood in all cases of vomiting where ulcers may be suspected, even when the superficial aspect does not indicate presence of blood.

The hæmorrhages may be arterial, venous, or capillary.[14] If in reference to the latter, it has been stated that, for instance, in cases of obstruction of the portal venous system, gastric ulcers ought to have been found frequently, we must remember that in those stages when blood extravasation from the vessels of the gastric mucosa occurs, probably all secretion of gastric juice has ceased for a long time.

It cannot be astonishing that in a morbid process which, as to its expansion to breadth and depth, is subject to the most variable conditions, the clinical pictures, too, vary to a great extent. Besides the typical forms, hazy atypical clinical pictures of symptoms may be found, which even the most experienced clinician cannot recognise. We, therefore, consider it our task to discuss especially not only the typical forms of ulcers, but also the atypical ones.

Diagnosis.

In the *diagnosis* of "gastric ulcer" the following *subjective* symptoms must be considered: gastric or vertebral pain, condition of the appetite, vomiting and the evacuation of blood by the rectum (melæna). *Objectively* important symptoms are development of the disease, pain or pressure in the epigastrium and dorsal region, condition of the stomach-contents and intestinal functions.

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[8] P. Hampeln, St. Petersburg medicin. Wochenschrift, 1891, No. 8.

[9] Pilliet et Deny, Gaz. medic. de Paris, 1893, No. 34.

[10] A. Fränkel, Deutsche medicinische Wochenschrift, 1894, P. 155.

[11] Dieulafoy, Exulceratio simplex, Bulletin de l'Academie de Médecine, 1898; Clinique médic. de l'Hôtel Dieu de Paris, Paris, 1897, Bd. II.

[12] [Rodman, Philadelphia Medical Journal, June, 1900. Table of interesting cases.]

[13] H. v. Hösslin, Münchener med. Wochenschrift, 1890, No. 14.

[14] [Rodman, *l.c.*]

(a) Subjective Symptoms.

The pain of the ulcer may be burning (sensation of "a sore spot") or stinging or cramplike. In the latter case it radiates from the epigastrium towards right or left to the vertebral column; it may also comprise the whole median and lower abdominal region, or may radiate toward the sternum, heart, or lung region, or even into the arms. Sometimes there is not severe pain proper, but a sensation of painful pressure. Female patients cannot tolerate a corset, and protect the tender gastric region with all kinds of more or less suitable measures (wadding, animal pelt, etc.). In some cases, the greatest intensity of pain is less in the epigastrium than in the back, and that either directly on or next to the vertebral column. Very often the pain is of lightning-like and lancinating character in permeating from the epigastrium diametrically toward the back.

The pain in *typical* cases is connected with the ingestion of food and is closely bound up with the quality and quantity of food. Pain, however, may occur, too, on an empty stomach and during night-time, but usually with less intensity. The influence of the quality of foods upon the occurrence and intensity of pain is demonstrated by the fact that thin fluid substances pass the stomach without inciting severe pains, while solid foods cause an attack of gastralgia. The temperature of foods and drinks is of special importance. Ingestion of abnormally cold or hot foods is just as often the direct cause of formation of ulcer as it is the cause of repeated attacks of gastralgia. [I saw two cases of bloody stool directly after the ingestion of ice cream soda.]

The dependence of the attacks of pain upon the ingestion of food is conditioned by the fact that they are *synchronous* with the same or follow after a longer or shorter time. The attack of pain usually begins about thirty or forty-five minutes after ingestion; if it *regularly* begins later (ninety to hundred minutes) after the last ingestion, it would indicate the place of the ulcer as being below the pylorus. If pain occurs regularly immediately after or still during eating, this factor would indicate the seat of the affection as being on the cardia or the lower section of the œsophagus.

The *attacks of pain* may last a varying time, but they cease spontaneously after the emptying of the stomach or after vomiting. The pain, though of great intensity, leads but seldom to symptoms of collapse, to the occurrence of cold perspiration, to chills, etc., as it usually is of relatively short duration. Changes in the position of the body (according to v. Leube, particularly position on the left side) increase the painfulness, while absolute rest, perfect abstinence from food, and application of heat decrease the same.

Appetite shows a condition of greatest variety; at times it is normal, at times increases, at other times is absent. We must distinguish between loss of appetite and sitophobia on account of the expected pains. Sometimes hunger is abnormally increased (hyperorexia) particularly in cases of chronic recrudescent ulcer. As a rule, the tongue is clean and the taste is normal, but there are exceptions.

The *vomiting*, the result of the spasms, occurs at the acme of the attack and usually means, similarly to that of cholelithiasis, the end. More or less large masses of usually well-digested ingesta which may be partially mixed with mucus, and are characterised by more or less strong acid reaction and abundant hydrochloric acid, are removed by the vomiting.

The *vomiting of blood* is by no means bound up with the paroxysm of pain, but, on the contrary, follows hours or even days after the same; it develops gradually, occurs in condition of apparent health, suddenly during the night, during sleep, in the early morning, or on a walk. According to Jaworski and Korczynski,[15] the acidity of the gastric juice is considerably increased shortly before and after hæmatemesis. The rapid transformation of oxyhæmoglobin into hydrochlorate of hæmatin is explained by this fact; the vomit becomes coffee-coloured, partial coagulation already occurs in the stomach. Only in cases of repeated and abundant hæmorrhages upon the gastric mucosa, the blood, in spite of the presence of an ulcer, may keep its particular colour and may, in the individual case, render the opinion about the origin difficult. Such a condition may easily occur in cases of coincidence of ulcer and phthisis of the lungs. As observations of this kind are rather rare, the following case may serve as illustration:

Ludwig P., well till two years ago; since that time cough, frequently bloody sputum. Several times hæmatemesis and tar-stools; simultaneously there exist painful pressure, nausea, eructation, with following vomiting. Patient lost 10 kilogrammes weight. Last hæmatemesis, November, 1888; previously, severe cramplike pains in the epigastrium. Status præsens: over apices, especially on right side, percussion sounds dull anteriorly and posteriorly; bronchial respiration, small râles, tubercle bacilli in sputum. Epigastrium very tender on pressure, upward to the upper third of the sterno-umbilical line. Stomach considerably atonic, greater curvature two fingers below navel. Stomach-contents show always *weak* colour reactions (resorcin, tropæolin, congo, phloroglucin-vanillin). Total acidity varies between 27 and 31.

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[15] Jaworski and Korczynski, Deutsche medicinische Wochenschrift, 1886, No. 47 to 49.

In reference to the distinction of a hæmorrhage from a peptic ulcer and other causes, see Section on Differential Diagnosis.

When larger hæmorrhages in the gastric surface have occurred, there will be usually evacuations of "tar-coloured" stools. The latter, however, may occur without a previous vomiting. In cases of severe hæmorrhages the evacuations may exhibit some unchanged blood. Frequently hæmorrhages by the mouth or rectum are preceded by marked symptoms of anæmia or even swooning. In doubtful cases the suspicion of intestinal hæmorrhages must be substantiated by the demonstration of blood-pigments (by the guaiac test*) or by the formation of hæmin-crystals. This examination may simultaneously be a guard against their being mistaken for similar pigments (iron, bismuth, huckleberry-pigment, wine-pigment, coffee, chocolate, etc.).

(b) Objective Symptoms.

Development of the Disease.—The development of the round ulcer of the stomach is protracted, as a rule, and the symptoms gradually reach the acme. In the beginning the painful pressure manifests itself only after heavy meals or foods not easily digested; subsequently the bill of fare that can be well borne becomes smaller and the selection more difficult, and the patient has to depend gradually on liquid foods. As the pains increase in intensity, the inclination to vomiting grows. Nevertheless, the general condition of the patient remains fair. Rest and bland diet are necessary conditions for the patient. As the first-named condition, as a rule, is not complied with at this stage of the disease, the process either remains unchanged, or a gastric hæmorrhage or evacuation of tar-coloured stools suddenly occurs, accompanied by symptoms of acute anæmia. Under favourable circumstances cicatrisation of the ulcer and final healing take place; in other cases, there may be a relapse; in still other cases there may be sequelæ and complications of various kinds.

Pain on Pressure.—We differentiate between an *epigastric* and a *dorsal* pain on pressure. The former, as a rule, makes itself felt on the median line, sometimes more to the left, rarely to the right of same. The level at which the pain exists depends on the situation and position of the stomach. Under normal conditions it is usually felt close to the processus xiphoides; in cases of gastroptosis it may be at a distance of several centimetres. Furthermore, it is evident that in changes of the situation of the stomach, especially the vertical one, extraordinary deviations from the usual seat of pain occur. In some cases we do not find an isolated point of pain, but a more or less extended area of pain; but even in such cases it is in the immediate proximity of the median line. The *dorsal pain on pressure,* to which I first called attention, is localised and more circumscribed than the epigastric one. It is generally located at the level of the tenth to the twelfth dorsal vertebra alongside of the vertebræ in a lateral expansion of two or three centimetres and a length of one to four centimetres. The pain on pressure in this case usually is on the *left* side, in some cases on both sides (in which case it is, as a rule, more intense on the left), and in rare cases on the right side only. Occasionally a point or an area of pain is found as far up as the fourth or fifth dorsal vertebra. The dorsal algetic point is present, according to my observations, in at least one-third of all cases of ulcer. In a few cases the painful tenderness is limited exclusively to this dorsal zone, and then is of greatest diagnostic and differentially diagnostic importance,* especially if the epigastric tenderness is not much marked. As an instance I refer to the following observation:—

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* Compare Part I, P. 224.

A young merchant, 25 years old, is referred to me by a physician with the inquiry whether an ulcer is present. The complaints of the patient consisted of occasional pressure in the epigastrium, which increases by the ingestion of food. No vomiting, particularly no bloody one, and no tar-stools. On examination no abnormal epigastric tenderness, but a marked circumscribed zone of pressure on the left side of the twelfth dorsal vertebra is found.

I made, therefore, the following diagnosis: Ulcer, probably on the posterior gastric wall. The patient underwent a successful cure of ulcer, and as he felt well, he indulged anew in over-eating and suffered a week later from a severe attack of hæmatemesis.

Pel[16] also found the epigastric point missing in cases of ulcer of the posterior wall, while the dorsal point was present in a characteristic manner.

The epigastric point is characterised not only through its marked localisation, but through its intensity. The latter differentiates it from all other possible forms of pain in the stomach. The patient makes a wry face even on regular easy passes over the epigastrium, the same occurs also on tapotement of the stomach (a diagnostically important symptom). Measured with the *algesimeter* described by me, the algesimetric value in the area of the maximum tenderness is from two to (utmost) four kilos. *Values above four kilos speak rather against the presence of a peptic ulcer.* The dorsal algetic point also shows, in comparison to the surroundings, an algesimetric value *of five to six kilos.*

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* Kuttner (Lindner-Kuttner, Chirurgie des Magens, Berlin, 1898, P. 119) does not consider the dorsal pressure-point as a diagnostically decisive symptom, as it is rarer than the epigastric pressure-point, and even when present frequently changes its location. I need not call attention to the fact that I consider only such cases as positive in which the painful area is constantly present at the same spot. This dorsal tenderness is not found constantly; but when present, according to my view, it is a valuable symptom, as far as my experience goes.

[16] Pel, Ebstein-Schwalbe, Handbuch der praktischen Medicin, 1900, Bd. 2, P. 502.

Vomited Material and Stomach-Contents.—In some cases the examination of the vomited material alone will show the increase of hydrochloric acid which is frequently found in cases of peptic ulcer; but, as a rule, the vomited material is mixed with so much mucus that the pathologic character of the same may often be hidden. The application of the stomach tube in cases of suspected ulcer is not without possible danger, as emphasised in General Part (page 103). In my opinion, it must not be applied under any condition if plain symptoms of ulcers are present. If there are doubtful symptoms of an ulcer, and either no recent hæmorrhage or none at all has occurred, it may be likewise preferable to avoid an introduction of the stomach tube; but a careful attempt at exploring the stomach may be permissible. [D. D. Stewart as well as myself has frequently passed the stomach tube in cases of gastric ulcer without the least possible danger—not so much for diagnostic as for therapeutic purposes in the application of solutions of silver nitrate.] The examination of the stomach-contents in cases of gastric ulcer has frequently shown an increase of the production of hydrochloric acid, occasionally up to three, four, or five per mille. It is an indisputable service of Riegel[17] and his disciples to have called attention to this occurrence. Later v. d. Velden, Korczynski, Jaworski, Gerhardt, Ewald, Rosenheim, and others have confirmed Riegel's statements.

The *intestinal functions* are sometimes normal, but more frequently sluggish, there is more or less obstinate constipation, [as in the majority of all gastric affections when hyperchlorhydria is present]. Sometimes stools cannot be evacuated without artificial measures. Where this is the case, only dry and hard scybala covered with mucus and blood are evacuated. Diarrhœa or alternate constipation and diarrhœa are, as far as I know, rare in cases of ulcer.

The seat of the ulcer may be guessed, in many cases, from the location of the algetic area, but cannot be definitely ascertained, for the size and position of the stomach are subject physiologically to great variations. Only ulcers of the cardia make an exception, as we emphasise in agreement with v. Leube.[18] The pain immediately following the ingestion of food and the excessive tenderness exhibited against otherwise normal temperatures of food and drinks allow in most cases the seat of the ulcer to be pointed out on the cardia. A subtile localisation, which, as a rule, is more or less hypothetical, is of no special value for the diagnosis of the ulcer;

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[17] F. Riegel, Zeitschrift für klinische Medicin, 1886, Bd. II, Heft 1, S. 12; Deutsche medicinische Wochenschrift, 1886, No. 52; Ueber Diagnostic and Therapie der Magenkrankheiten, Volkmann's Sammlung klinischer Vorträge, No. 289; Beiträge zur Diagnostik der Magenkrankheiten, Zeitschrift für klinische Medicin, 1887, Bd. 12, Heft 5; Die Erkrankungen des Magens, Wien, 1897.

but for the prognosis of the ulcer and possible surgical interference, it would be of greatest interest to have the seat of the ulcer satisfactorily ascertained. See Chapter on Treatment.

Diagnostic Importance of the Individual Symptoms.

The number of the particular and characteristic symptoms is small: Vomiting of blood in conjunction with previous attacks of cardialgia, even if objective examination was negative, is decisive in spite of a few exceptions. As the same, however, is absent in the first stages and in many types of ulcer, we are dependent sometimes solely upon the above-described characteristic ulcer pain. In the presence of this symptom alone ulcer or erosions may be diagnosed if it is pronounced in the above-described form.

The chronic course of the process (ulcus *chronicum*), as well as the characteristic algetic points, are the most important of the other objective symptoms. As to the former, it deserves to be emphasised that a chronic process must be assumed even in such cases where temporary, even occasionally long, intervals of relatively good gastric condition have preceded. If the characteristic subjective symptoms, *i.e.,* those particular gastric pains accompanying the ingestion of food, are decidedly marked and join the above symptoms, the diagnosis of ulcer is almost certain. According to my opinion, the value of examination of stomach-contents is much more uncertain. If the symptom-complex is otherwise plain, the examination becomes superfluous. Would anybody venture, if the complex is uncertain, to give decisive importance to the finding of increased secretion of hydrochloric acid, which, as is well known, may occur in numerous other gastric affections?

Diagnosis of the Atypical Form of Peptic Ulcer.

In some cases all symptoms of the chronic ulcer of the stomach may be absent until the case is explained by sudden occurrence of hæmatemesis or even of perforation. It is evident that cases of this kind usually cannot be diagnosed. Cases in which uncertain dyspeptic symptoms are prevalent are equally difficult of diagnosis. In such cases v. Leube's[18] suggestion may be utilised to put the affection to the test, so to speak, by the treatment recommended by the said author. It is true, a favourable result of such a treatment does not make the diagnosis of a gastric ulcer a certainty, but may make it probable to a certain extent. Observations of this kind are known to every busy practitioner, so that I need not mention any instances from my own practice. Furthermore, there are other forms of ulcers which vary so much from the above-described symptom-complex in its particulars that the diagnosis is sometimes impossible, or at least difficult. This is especially the fact in all cases where gastric hæmorrhage is absent. Above all, the most valuable diagnostic means rest in the seat and the manner of occurrence of pain. These symptoms also may be missing, and yet the further course may show the presence of an ulcer. Of course, in this respect, only such cases in which hæmatemesis occurred in the course of the disease are positive. I refer only to the following three cases:—

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[18] v. Leube, Specielle Diagnose, etc., S. 246.

Case I.—Mrs. E. K., widow, 43 years of age, hereditarily nothing known. Patient claims to have formerly always enjoyed good health. Between the twenty-fifth and thirtieth year of her life, frequent vomiting and gastralgia. In her thirty-eighth year, treatment for tape-worm. Since that time, appetite not good. For the last five weeks she has complained of sensation of pressure in the stomach (no pain), particularly while walking; distension in the epigastrium, weakness, and loss of appetite. Stools regular. Patient never complained of pain after meals; the kind of food did not influence the gastric disturbances. Two weeks ago sudden vomiting of partially brown masses, partially coagulated blood, with increasing weakness. Patient is not able to give any statement as to the condition of the stool after this occurrence. Examination shows, besides a coated tongue, as only symptom a marked tenderness in the epigastrium (algesimeter 2.5 kilos), and on the left, posteriorly, between the tenth and twelfth dorsal vertebræ (algesimeter, 7.5 kilos).

Case II.—Anna T., seamstress, 23 years of age; father died of phthisis pulmonum; mother, sisters, and brothers well. From her sixteenth to twentieth year patient was very anæmic; first menstruation in her eighteenth year. For years there were periodically occurring attacks of headache and vomiting. She vomits particularly after greasy foods and milk; otherwise she can eat any food. Vomiting occurs frequently after exertion. Patient vomited blood several times in the year 1892, the last time eight weeks ago. Since that time, severe stinging pains in the epigastrium and the small of the back. *The same are not severer after meals than on empty stomach.* Appetite good, constipation, menstruation regular. Nothing found except tenderness in epigastrium, resistance (adhesive peritonitis?) and marked tenderness in the right parasternal line, no dorsal pain.

Case III.—L., laborer, 41 years of age. Patient enjoyed good health until his present complaint. Suffering from gastric disorders for the last six years, beginning with regurgitation of sour-tasting fluid up to the mouth. Drinking denied. From the onset vomiting of reddish-brown liquid blood, at the same time blackish stools. The hæmatemesis reoccurred about every half year. Appetite always good. *Patient could tolerate food of any kind.* Only occasionally stinging pains in the gastric regions, independent of meals, radiating toward the vertebral column. Status præsens shows normal borders of liver and spleen, a circumscribed tender spot in the epigastrium (algesimeter, 2 kilos), dorsal algetic point on the left side of the twelfth vertebra.

Furthermore, I observed several cases of gastric ulcer in which the attacks of pain occurred periodically as in cases of neurosis. The following two may be mentioned:—

Case IV.—Miss R., 25 years of age. Menstruation regular since thirteenth year. All of her five sisters and brothers have stomach-complaints, and are more or less nervous. Patient herself, though suffering for years from heartburn, had good appetite and could stand all kinds of food. In October, 1890, patient had severe attack of influenza; since that time vomiting and severe pain in the gastric and intestinal regions, anorexia. Bowels were very much constipated, and only opened after taking medicine. Patient was confined to bed on account of great emaciation. In January, 1891, tar-like stools occurred after pains had increased to unbearableness. A few days later severe peritonitis, so that patient was unable to move in bed on account of pain. Typical treatment for ulcer. Decrease of pain, gradual recovery. After leaving the bed, renewed vomiting and pain, even after the blandest food. As a whole, the picture was changeable. *Sometimes food could be borne without pain, at other times it could not.* Appetite increased gradually, bowel movement became slowly regular. The clinical picture is at the present time of such a character that, in spite of one and the same method of living, after entirely normal digestion lasting for a few days, suddenly pains reappear, which again disappear spontaneously. Present state shows intense tenderness close to the sternum (algesimeter, 0.75 kilos), posteriorly, on the left of the fourth dorsal vertebra. Localised tenderness (nervous pressure-point?), otherwise nothing abnormal.

Case V.—G., engineer, 40 years old, suffered for the last three years from gastric disturbances. He complained particularly of occasional cramp-like gastric pains, not dependent on the ingestion of food. Appetite in the beginning good; stools regular. The discomfort increased gradually till in March, 1891, *characteristic hæmatemesis and tar-stools appeared.* The following summer, after Carlsbad cure, improvement, but only of short duration. Attacks of pain repeated themselves periodically every two to four weeks; they lasted about one or two hours, and occurred in spite of the most careful diet. Appetite is deficient; patient lost about 10 kilogrammes of weight in the last three years. Present state shows normal condition of respiratory and circulatory apparatus, urine normal. Epigastrium very tender close below the ensiform process to an extent of 3 centimetres. No dorsal algetic point.

It is noteworthy that in all five cases a marked painful tenderness on pressure, and in two of them dorsal algetic points, were present.

A particular source of mistaken diagnosis rests in the fact that symptoms of nervous dyspepsia are found together with undoubted symptoms of gastric ulcer. The following observations may be mentioned:—

Miss E. G., employee, 22 years of age. Patient stated that she suffered from occasional gastric pain for several years past. One and a half years ago, pleurisy on left side. For the last three weeks, continuous pains and vomiting. The former begin early in the morning, before partaking of food, and end by vomiting mucus. After meals the pain increases, at the acme of the pain vomiting bringing relief. Milk is well borne. Bowels irregular, constipated; loss of weight in the last five weeks amounts to 7 kilogrammes. Present state: pale, but strong girl, with strongly-developed bones and good development of adipose tissue. Mucous membranes anæmic. Heart and lungs normal. Abdomen not relaxed. Epigastrium, particularly 3 centimetres below the ensiform process, intensely tender on pressure. At the level of the tenth dorsal vertebra, marked algetic point.

Treatment: The same consisted first of milk diet and solution of nitrate of silver, which, however, was not well borne. In spite of this, improvement, in so far as no pain or vomiting occurs on an empty stomach, but only after ingestion of food. In July, 1899, v. Leube's rest-cure, with good result. After returning to her occupation in October, 1889, renewal of pain and vomiting. On examination of the stomach after test-breakfast, normal condition of secretion and acidity was found; after test evening meal, the stomach was found empty in the morning. November, 1899, repeated v. Leube's treatment for ulcer. No effect. As the proposition of absolute rectal feeding was not accepted, patient was referred to the City Hospital at Urban for an operation, beginning of April, 1900.

There a nervous affection was suspected; consequently treatment with lavage, diet, electric light bath. Nevertheless pain and vomiting continued. On April 23, 1900, gastro-enterostomia retrocolica (according to v. Hacker) by Professor Körte. Stomach was considerably dilated. Serosa of the pylorus somewhat thickened. Lumen of the pylorus somewhat stenotic. Excellent condition; all disturbances cease. On April 24, 1900, transfer of the patient to Blankenberg, into the Home for Convalescents. Four weeks later, after excitement, reoccurrence of pain and vomiting, and subsequent readmission of the patient to the hospital. As a vicious circle is suspected, entero-anastomosis is performed. Nevertheless, in the first few days vomiting continues, but ceases after patient was isolated. From that time till now (October, 1900), very favourable course. Patient does not suffer any longer; has gained 11 kilogrammes in weight since the first operation. No painful tenderness in the epigastrium nor in the back.

Epicrisis: Without doubt in this case organic changes (slight stenosis of the pylorus, thickened serosa of the pylorus) are present, which prove a former ulcer. Moreover, a special hyperæsthesia of the gastric mucosa must be assumed, as shown by the course of the case.

Differential Diagnosis.

In the differential diagnosis between gastric ulcer and other affections it is a matter of greatest importance whether the history of the case shows gastric hæmorrhage or bloody stools or not. In the former case it is necessary to decide whether the bleeding came from the stomach, and if so, whether it originated in an ulcer or whether it came from some other source.

1. *Does the blood originate in the stomach?*

Hæmorrhages from the gums, pharynx, or nasal cavity usually do not make the differential diagnosis difficult on careful observation; but hæmorrhages from the lungs on one side and hæmorrhages from the œsophagus on the other side, may cause mistakes. The following characteristics may serve to differentiate between gastric and pulmonary hæmorrhages:

Pulmonary	Gastric
Hæmorrhage	
1. Blood is light red, foamy.	1. Blood is dark brown, partly coagulated, frequently mixed with food-remnants.
2. History points to a pulmonary or cardiac affection (hæmorrhagic infarct).	2. History points to symptoms of either gastric affection or stasis in portal circulation.

3. Physical signs point to a pulmonary or cardiac affection—the stomach may be affected secondarily.	3. Physical examination evinces a gastric or hepatic affection, or stasis in portal circulation (tumours, thrombosis).
4. Pulmonary hæmorrhages, followed by rusty-coloured sputa for days (there are exceptions); no blood in the stools.	4. Gastric hæmorrhages are frequently associated with tar-coloured stools.

The above-named characteristics are usually sufficient to ascertain the origin of the blood. But as cases of pulmonary hæmorrhages may be complicated with gastric hæmorrhages (for instance, case, page 430), the differential diagnosis may be very difficult under certain circumstances.

Differentiation of gastric and œsophageal hæmorrhages is often quite difficult. In cases of œsophageal bleeding the following causes must be considered: cancer, ulcer, broken varicosities, passive congestion. The examination of the œsophagus (carcinomatous stenosis) or the demonstration of an affection producing stasis of the venous circulation (above all, cirrhosis of the liver) would be of great diagnostic importance. The character of the blood itself is different, as in cases of œsophageal hæmorrhages the blood is dark red but not brown, and is usually not mixed with food remnants.*

Some part of the blood, however, may reach the stomach and intestine and cause coffee-ground coloured vomiting and tar-coloured stools. The fact that in cases of œsophageal ulcer pain occurs immediately when the food passes through the œsophagus, and that in cases of gastric ulcer pain follows only some time after the meal, may be of great importance in the differentiation between œsophageal and gastric hæmorrhages. Furthermore, the pain directed from the sternum towards the epigastrium radiates towards the shoulder-blades or the back. Retching and vomiting are mentioned in all cases of ulcer of the œsophagus.

Age and sex may be of differential diagnostic value also, as œsophageal ulcer in contrast to the gastric ulcer is said to occur particularly in men beyond the fortieth year of life. Differential diagnosis between the two forms is considerably interfered with by the fact (Quincke, Debove, Zahn, and others) that œsophageal ulcer may be associated in rare cases with gastric or duodenal ulcer.

2. *Does the gastric hæmorrhage originate in an ulcer or in some other affection?*

Besides ulcer, we have to consider carcinoma, portal vein stasis, gastric varicosities, trauma, scurvy, hæmophilia, leukæmia, Hodgkin's disease, acute yellow atrophy of the liver, ruptures of blood-vessels, acute infectious fevers, such as typhoid, yellow fever, cholera, plague, malaria (Hemmeter), acute and chronic gastritis (ulcerosa), and pyloric stenosis; miliary aneurisms, corrosion through caustic poisons; syphilitic and arteriosclerotic conditions; hysteria, cholelithiasis, etc. The hæmorrhages of gastric carcinoma are usually easily differentiated from those of ulcer. The former are, as a rule, small in quantity, they do not bring about bloody stools, or at least very rarely; the blood is decomposed and of a coffee- or chocolate-brown colour. Cases of carcinomatous ulcers (compare Chapter on Cancer of the Stomach) and carcinoma of the cardia, in which severe hæmorrhages occurring towards the end may terminate fatally, are exceptions. In rare cases of hysteria and crises gastriques (vomissement noir, Charcot) hæmatemesis has been reported. These instances are very rare, and the picture is essentially different; in such cases, however, organic or functional nervous diseases may be coincident with ulcer, as Debove rightly emphasises.

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* But there are notable exceptions. Only a short time ago I treated a patient suffering from carcinoma of the lower section of the œsophagus, who repeatedly vomited brownish blood mixed with food. This food came out of the diverticle above the stenosis.

Hæmorrhages resulting from stasis in cases of liver affections, especially cirrhosis, may easily be mistaken for hæmorrhages from gastric or duodenal ulcers. Leichtenstern[19] pointed out that there is a form of cirrhosis of the liver which may give almost the precise picture of the course of an ulcer. Repeated, usually very copious, gastric hæmorrhages, which return in intervals of weeks and months, take place. The patients are very anæmic, not at all jaundiced, ascites and enlargement of the spleen are absent, probably on account of considerable hæmorrhages, through which the portal circulation is relieved. Leichtenstern found a latent cirrhosis of the liver in several cases of this kind, which terminated fatally within a few days from repeated gastric hæmorrhages.

Stein[20] recently described a very interesting case of cirrhosis of the liver which was diagnosed as a duodenal ulcer until ascites made its appearance.

We should always, therefore, think of the possibility of a developing cirrhosis of the liver in all cases of sudden gastric hæmorrhages of persons who had been healthy up to this time, and the more so if symptoms of a gastric ulcer have not preceded or followed a hæmatemesis (Oppolzer). Gastric hæmorrhage or melæna occurs in cases of cholelithiasis with or without icterus and may suggest gastric ulcer, particularly as other subjective or objective symptoms may be entirely absent at this stage or may be masked.

Differential diagnosis may be often difficult in cases where hæmorrhages occur suddenly and previous history does not give any hints. Decrease of liver dulness and enlargement of spleen may be decisive, though their absence does not exclude cirrhosis of the liver, as just mentioned. Where previous history is known, we have on the one side to pay attention to the presence of epigastric painfulness, which is either missing entirely or, if present, is only slight, and on the other side syphilis or abuse of alcohol.

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[19] Leichtenstern, Penzoldt-Stintzing's Handbuch der speziellen Therapie, 1896, Bd. 4, S. 141.

[20] Stein, Archiv für Verdauungskrankheiten, 1899, Bd. 5, S. 77.

Besides the above-named sources, there are, as described by Harttung,[21] Hampeln,[22] Ewald,[23] Reichard,[24] and Dieulafoy,[25] certain parenchymatous hæmorrhages, for which Harttung suggested the name of "plica-hæmorrhage," as they usually originate in the folds of the gastric and intestinal mucosa. In these cases the gastric mucosa may be perfectly intact, with the exception of small erosions, visible sometimes only by the magnifying glass. Dieulafoy recently described these occasionally fatal ecchymoses, which affect only the most superficial strata of the gastric mucosa, as "exulceratio simplex," and sees in them the first stage of the true Cruveilhier's ulcer.

Furthermore, gastric hæmorrhages may occur as a result of *hæmophilia;* the following case may serve as an instance:—

A. H., merchant, 38 years old, comes from a hæmophilic family, and that on the maternal side. Mother, who is 72 years of age, suffered frequently from severe menorrhagia as well as bleedings from her gums; only a short time ago there was intestinal hæmorrhage without definite cause. A sister of the patient, 40 years of age, had, after birth of a child, a most perilous uterine hæmorrhage, which finally, after almost everything had been tried, ceased after hot irrigations. Since that time, weak and nervous, she must always stay in bed for two days during the perilous menstruation. A brother of the patient, 38 years of age, suffered always from severe epistaxis, and was operated on for goitre by Professor Sonnenburg in 1886. On account of abundant bleeding, goitre was only partially extirpated. Five years later blood-poisoning (pyæmia?) from the wound, as is claimed, which terminated fatally. Another sister, 35 years of age, is very chlorotic and nervous; loses blood through the stool at times.

Patient himself had typhoid fever in his sixteenth year; three years later, articular rheumatism. Ten years ago, after extraction of a tooth, severe bleeding, so that tampon had to be used. The first gastric hæmorrhage occurred in 1885; copious hæmatemesis and tar-coloured stools were preceded by weakness, prostration, and nausea. *Except this, neither before nor afterward any digestive disturbances, particularly never epigastric or dorsal pains.* Patient could, and can tolerate, the heaviest meals without any trouble.

The second hæmorrhage occurred in 1887 while patient was in the best of health; patient vomited large quantities of light-red blood, and again tar-coloured stools followed. Confinement to bed for a week to ten days; slow convalescence.

Since that time bleeding was repeated six times, the last time only from the intestines; the last hæmorrhage of moderate degree occurred in December, 1892.

The careful examination of the perfectly well-looking patient does not at all point to any gastric or intestinal affection; no painful tenderness in the epigastrium or duodenal or dorsal region.

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[21] O. Harttung, Deutsche medicinische Wochenschrift, 1890, No. 38.

[22] Hampeln, St. Petersburg medicinische Wochenschrift, 1891, No. 8.

[23] Ewald, Eulenburg's Realencyklopädie, 3 Aufl., Bd. 14, S. 288.

[24] Reichard, Deutsche medicinische Wochenschrift, 1900, No. 20.

[25] Dieulafoy, Clinique médicale de l'Hôtel Dieu, 1897-98, S. 1 *et seq.*

According to observations of L. Kuttner[26] there frequently occur in chlorotic persons the so-called *menstrual gastric hæmorrhages,* which periodically take place in disturbances of the menstrual functions. Kuttner refers these formerly so-called "vicarious" gastric hæmorrhages to increased congestion of the blood-vessels, which, particularly in chlorotic persons, exhibit a diminished resistance (fatty degeneration, etc.).

As to differential diagnosis, other diseases have to be considered: cholelithiasis, chronic gastritis (especially that accompanied by atrophy of the gastric mucosa), gastralgia, nervous dyspepsia, enteroptosis, cancer, and colitis.

1. Usually only such cases must be considered in the differential diagnosis between *cholelithiasis* and *gastric ulcer,* in which, on the one hand, the characteristic hæmatemesis and tar-coloured stools are absent; on the other hand, biliary grit or sand or gallstones are not observed. In such cases the following points are of importance:—

(*a*) *Pain.*—The same in cholelithiasis is not, or at least not solely, in connection with the ingestion of food, and occurs, for instance, even if the diet is very bland (milk, tea, soups), in equal intensity as when the food is difficult to digest; usually it draws from the median line or from the right side of the same towards the hepatic region; the presence of the dorsal pain to the right of the vertebræ at the level of the twelfth thoracic vertebra, two or three fingers' breadth from the vertebra. (Compare General Part, page 62.)

(*b*) *Hepatic Enlargement.*—In cholelithiasis the right lobe of the liver (more seldom the left lobe) is enlarged for a short time after an attack, and is markedly tender (bimanual palpation); the gall-bladder and gall-bladder region are equally tender.

(*c*) *The Course.*—In cholelithiasis there is well-being and tolerance to all kinds of food, alternating with periods of paroxysms of pain, which may be occasioned by even the slightest nourishment. Similar intervals, it is true, may occasionally be observed in ulcer of the stomach, but they are very rare.

(*d*) *Examination of Stomach-Contents.*—In ulcer, hyperchlorhydria is a frequent symptom, as mentioned above, while in cholelithiasis the amount of HCl is either normal or deficient.[27]

(*e*) *Jaundice.*—If jaundice is repeatedly observed following attacks of pain, it would speak against ulcer of the stomach, but not against duodenal ulcer; with the latter icterus has been occasionally observed.

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[26] L. Kuttner, Berliner klinische Wochenschrift, 1895, No. 7.

[27] J. Leva, Virchow's Archiv, 1893, Bd. 132, P. 490.

(*f*) *Fever.*—Fever occurs not infrequently as a complication in cholelithiasis and cholecystitis. In ulcer it is exceptional, perhaps in cases of subphrenic abscess, perforation into the lungs, inflammatory adhesion of an ulcer of the fundus with the spleen, etc.

Finally, we must not forget that gastric ulcer and cholelithiasis may be coincident, as demonstrated by the following case:—

Mrs. Marie V., 37 years old, has been chlorotic since her youth; complains of pain on the right of the stomach, at times in connection with meals, at other times independently. Appetite always good. Later on in good health for six years. Since January, 1892, without special cause pain on the right of the stomach, usually about one hour and a half after meals. Ingestion of heavy or light foods is without influence as to the intensity of the pain; pain increases on walking; pain also on an empty stomach, but of less duration and intensity. Appetite good, but limited from fear of eating. Slight emaciation, considerable constipation. No nausea, no vomiting, no hæmatemesis, no icterus after the attack of pain. Lavage, performed elsewhere, without effect.

Physical examination shows marked enlargement of the very tender right lobe of the liver for 3 centimetres below the costal arch. The epigastrium is equally tender. No dorsal pain. Diagnosis of cholelithiasis made. Treatment with Carlsbad Mühlbrunnen and suitable diet, simultaneously fæces carefully examined for gall-stones; gall-sand was repeatedly found (test for cholestearine positive). After Carlsbad "cure," temporary improvement. In February, 1892, renewed complaints of loss of appetite and pressure in the epigastrium. The hepatic region was still very tender on palpation. In the beginning of March, 1892, suddenly colicky pain in the epigastrium immediately following meals, and terminating by vomiting at the acme. Pain was even present after ingestion of milk and other liquid nourishment. Pain was localised centrally in the epigastrium; at the same time distinct algetic point posteriorly on the left, between the twelfth thoracic and the first lumbar vertebra; no painfulness on the right side; hepatic region moderately enlarged and tender. Treatment: rest-cure lasting four weeks, hot poultices, liquid diet. No other medication. Already at the end of the first week, stomach and region of the twelfth thoracic vertebra is no longer tender on pressure; no attacks of pain. After four weeks, perfect well-being; after eight weeks, patient ate all kinds of food with impunity. Gain in weight, 4.5 kilogrammes. Stomach on pressure, without painfulness (algesimeter 6 kilogrammes); only the right lobe of the liver is still slightly tender on pressure. Patient has been treated by me repeatedly for recrudescent gastric pain in the course of the last three years.

2. The character of pain is especially decisive for the differential diagnosis between *gastric ulcer* and *atrophy of the mucosa*. In the latter cases there are paroxysms of pain, not connected with the taking of food. The algetic points so characteristic of the peptic ulcer are not present. Stomach-contents show absolute loss of HCl and ferments, a fact which is very rare in ulcer.

3. The following factors are of value in the differential diagnosis between *ulcer of the stomach* and *cardialgia*:—

(*a*) *Character of the Pain.*—Pains in cardialgia are not dependent upon the ingestion, occur with great severity, but periodically and without any visible connection with the nourishment; algesimetric examination, not made during the attacks, yields either normal tenderness in the epigastrium (6 to 9 kilos) or algetic points in a few places (plexus solaris, cœliacus, hypogastricus) with a tenderness which is smaller if compared with those in ulcer (4 to 6 kilos). In neuralgias, pain during the attack seems to decrease by pressure, but this symptom is seldom of diagnostic importance.

An essential, clinically important form of gastralgia is, according to my experience, represented by that accompanying anæmia and chlorosis.[28] It is differentiated from gastric ulcer by the independence of the attacks from the ingestion of food, by the long periods of entirely normal state of health, finally by the uselessness of a regular treatment for ulcer. Occasionally, however, I observed cases which made a differential diagnosis very difficult. (In the Chapter on Gastric Neurosis, we shall revert to gastralgia in anæmia.)

(*b*) *Presence of predisposing diseases,* particularly hysterics, anæmia, uterine and ovarial affections, poisoning (bromine, arsenic, antimony, [lead], etc.)' would point to the diagnosis of cardialgia or enteralgia.

It may be mentioned incidentally that in the course of a gastric ulcer, or after its cure, genuine neuralgias, especially *intercostal neuralgia,* not infrequently occur. Traube suggests their occurrence as dependent from irradiation of the sensible vagus fibres of the stomach. In a case reported several years ago by Eisenlohr,[29] neuralgia was caused by a direct irritation of the intercostal nerves through a large ulcer of the anterior gastric wall. The nerve medulla of the intercostal nerves showed a high degree of degeneration.

4. Observation during some length of time is necessary for a differential diagnosis between *gastric ulcer* and *nervous dyspepsia.* We must not fall into error through the presence of a circumscribed pressure point in the epigastrium. However valuable this symptom may be, it may lead, by too much emphasising the same, to wrong conclusions. On frequent and repeated examination it will be found that the painful area changes its seat within a few days. Previous history is also of importance, because in nervous dyspepsia it will show absence of hæmatemesis and bloody stools, the occurrence of sudden changes from excellent gastric function to severe gastric pressure and painfulness, no evident dependence of the latter upon quantity and quality of the ingesta. If all diagnostic methods fail, a regularly instituted treatment for ulcer may be the best test.

5. Differential diagnosis between *gastric ulcer* and *enteroptosis.* A

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[28] Boas, Zeitschrift für praktische Aerzte, 1897, No. 6.

[29] Eisenlohr, Aerztlicher Verein zu Hamburg, Deutsche medicinische Wochenschrift, 1888, No. 44, S. 910.

frequent accompaniment of enteroptosis which will be fully discussed below, is given in more or less marked algetic points, which are found usually a few centimetres above the navel. As in enteroptosis with or without nephroptosis, dyspeptic disturbances (particularly pain in various places of the abdomen) usually occur, differential diagnosis rests between enteroptosis alone and gastric ulcer complicated with enteroptosis. I had a chance to observe several cases of this kind; one of them may be reported in brief:—

Mrs. N., widow, 46 years of age, had two miscarriages. Syphilis not proven. Twelve years ago, for the first time, occasional gastric pain, but every kind of food was well borne. During the five years after the last miscarriage, continuous gastric pain, and that on an empty stomach as well as after meals. Pain ceases during night-time. Pain not more severe after ingestion of less digestible food, such as brown bread or potatoes. Pain radiates from the epigastrium toward the lower abdomen and in the back up to the shoulder-blade. Appetite deficient, bowels constipated, move only after taking purgatives. No nausea, no vomiting of water. Two years and a half ago, for the first time, hæmatemesis (about half a chamber-pot full), accompanied by severe pain in the epigastrium and weakness. Stools were tar-coloured for a few days following. Improvement after two weeks, but pain still present. A year later, again hæmatemesis of the same amount as the first time. Attack of fainting. Rest in bed, pills, powder, improvement. Four months ago, inflammation in the abdomen, from which patient recovered after an illness of two weeks. The gastric and abdominal pains began anew. Ingestion of food did not influence occurrence or severity of the pains. Physical examination showed ptosis of the right kidney (second degree, according to Ewald), gastroptosis. Epigastrium very tender. Algesimetric examination in epigastrium yields a painfulness of 3 kilogrammes, in the left parasternal line 2 kilogrammes. Posteriorly on the left of the transverse processes of the tenth to the twelfth thoracic vertebræ, and along the left twelfth rib likewise marked tenderness on pressure. Algesimetric test shows 4 kilogrammes; on corresponding right side, 10 kilogrammes.

This case proves that a tenderness in the epigastrium must by no means always be considered as nervous pressure pain, but that always a combination with an ulcer ought to be kept in mind. In such cases also the examination of the vertebral pain seems to me to be diagnostically important. It is true, we very frequently encounter in enteroptosis pressure points in the neighbourhood of the vertebræ, but they are located on diverse areas. A localised painful tenderness in the region of the tenth to twelfth vertebra, or, when the stomach is situated low, the first lumbar vertebra on the left side, points with some probability to a combination of enteroptosis with ulcer. On the other hand, it need not be discussed that absence of vertebral pain does not exclude ulcer.

6. The following points of view are important for the differential diagnosis between *ulcer* and *cancer of the stomach*:—

(*a*) *Course.*—In gastric ulcer, the course may extend for years, with remissions and exacerbations; strength, as a rule, remains comparatively good. Gastric ulcer is an affection of the younger and middle age, while cancer usually appears later in life, with a rather acute onset (v. Leube[30]), and its duration is seldom longer than two years. It is true, there may occur cancers of the stomach in younger years (one case under my observation was that in a girl of 27 years, another that in a young man of 18 years); there may, on the other hand, be an exceedingly debilitated condition in cases of simple gastric ulcer.

(*b*) *Pain.*—The same is usually independent of the ingestion of food, may be absent for days, and recur again very severely; *a particular type cannot be demonstrated.* Gerhardt[31] remarks, in his classic discussion on gastric ulcer, "Where the fear of pain forces a restraint of ingestion of food, one has to deal hardly with cancer, far more with ulcer, perhaps with cardialgia." The stomach is, as a rule, not as tender as in ulcer; yet there may be exceptions.

(*c*) *Vomiting.*—In ulcer, vomiting designates the acme of the pain. In cancer, however, it is irregular and occurs usually after a greatly protracted retention of the stomach-contents. As to the character of hæmatemesis in cancer, compared with that in ulcer, the subject has already been discussed.

It has been mentioned that in rare cases of cancer of the stomach, particularly carcinomatous ulcer, hæmorrhages take place which cannot be distinguished from those in simple ulcer. Cases of this kind may make considerable diagnostic difficulties, as I know from my own experience.

(*d*) *Stomach Contents.*—In gastric ulcer, the stomach-contents contain, as a rule, hydrochloric acid and no fermentative products, as long as dilatation has not ensued as a result of stenosis of the pylorus or spasmodic (dynamic) contraction of the pylorus. In cases of cancer of the stomach, hydrochloric acid is very frequently absent, but *the contents are usually intensely acid; the acidity, however, is distinguished by a great amount of lactic acid or lactates.* On microscopic examination, a peculiar type of bacilli (see chapter on Cancer) is found, which are not present in cases of gastric ulcer.

(*e*) *Tumour.*—Tumours in the stomach, especially in the pyloric section, may make their explanation very difficult, and have yielded, as shown by the increasing experience of the surgeon, equally frequent unpleasant as well as pleasant surprises. Errors may be occasioned by cicatrised thickenings at the base of an ulcer, encapsulated peritonitic exudates, hypertrophy of the pylorus, or a tumour in a neighbouring organ, gastroliths.

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[30] v. Leube, Specielle Diagnose der inneren Krankheiten, Leipzig, 1889, S. 252.

[31] C. Gerhardt, Deutsche medicinische Wochenschrift, 1888, No. 18.

Only the most careful consideration of all the symptoms in question will serve to guard against mistakes.

(*f*) *Œdema.*—Œdema occurs, it is true, in all diseases accompanied by severe cachexia to a greater or lesser degree; yet it is found so frequently in cancer of the stomach, at least in later stages, and, on the other hand, it is absent so frequently in gastric ulcer, that this symptom may likewise be turned into account for the differential diagnosis.

7. The differential diagnosis between *gastric* and *duodenal ulcer,* is more of scientific than practical importance. First of all, the typical ulcer pain occurring constantly a considerable time after the meal (two to four hours), tar-coloured stools without a simultaneous hæmatemesis and constant pain to the right of the median line, speak for duodenal ulcer.[32] Second, the following conditions are to be considered: in duodenal ulcer, icterus occurs in rare cases; in gastric ulcer, it never occurs. If the ulcer is situated in the stomach, dyspeptic disturbances, especially vomiting, prevail; in duodenal ulcer they are exceptional. Only where the first-named symptoms are present, we may diagnosticate duodenal ulcer with some degree of certainty, while in other cases diagnosis can be considered only as a probable one.[33]

8. Differential diagnosis between *gastric ulcer* and *affections of the colon.*—According to Schütz,[34] with whom I agree in all essentials, affections of the colon, especially colitis and enteroptosis, may closely mimic the clinical picture of gastric ulcer. Careful inspection of the stool on the one side, and examination of the stomach-contents on the other side, will frequently establish the differential diagnosis. From my own experience I would emphasise that in many cases of colitis, painfulness is found not only in the transverse colon, (which, by the way, is never localised so strictly as in ulcer), but also in other sections of the colon, and that this fact will help to make the diagnosis certain.

Diagnosis of the Complications and Sequelæ of Gastric Ulcer.

We mention in the following only the most important complications and sequelæ of the round ulcer of the stomach, as a few have only a purely casuistic importance, and may therefore be omitted. In the order of frequency, the following complications of the chronic gastric ulcer may be considered:—

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[32] *cf.* H. Oppenheimer, Das Ulcus pepticum duodenale, treated especially from the ætiologic-statistic standpoint. Diss.-inaug., Würzburg, 1891. (There the literature of Ulcus duodenale.)

[33] Compare also Boas, Diseases of the Intestines, New York, 1901, P. 289; [Bernheim, Pennsylvania Medical Journal, February, 1906].

[34] Schütz, Verhaundlungen der XVII Krongresses für innere Medicin, 1899.

(1) Cicatricial stenosis of the pylorus. (2) Perforation in peritonitis. (3) Perigastritis. (4) The transition of ulcer to carcinoma (carcinomatous ulcer). (5) Hour-glass stomach. (6) Subphrenic abscess.

1. *Stenosis of the pylorus* is the most frequent complication of all gastric ulcers situated at the pylorus and its immediate neighbourhood. Stenosis of the pylorus may be manifested in a short time (in a few months). As the diagnostic symptoms of stenosis of the pylorus coincide with those of gastric insufficiency, they will be discussed in the next chapter.

2. *Perforation Peritonitis.*—According to Gerhardt,[35] perforation of a gastric ulcer takes place in thirteen per cent. of all cases; according to Habershon,[36] even in eighteen per cent. The symptoms of perforative peritonitis, especially in cases of gastric ulcer, manifest themselves in the following manner. Uusally in apparent health, certainly without any alarming prodromal signs, there develops a very grave, rapidly threatening clinical picture. The patient suddenly feels a severe, unbearable pain, under the intensity of which he immediately collapses. In a very short time the pulse becomes small and frequent, and later filiform; the extremities become cold and numb; cold sweats and facies Hippocratica complete the terrible picture. The abdomen may be painful on pressure, even at the slightest touch, but this symptom is not always present; there may be various degrees of meteorism, from the slightest, just visible, distension to the most extreme stages of tympany, with disappearance of liver and spleen dulness. But—and this is diagnostically of the greatest importance—*meteorism is by no means a regular accompaniment of the perforation of ulcer, just as it is not in cases of perforation of a suppurated appendix.* In ulcer, several factors evidently play their part. First, whether the perforation ensued when the stomach was empty, whereby the point of perforation closes readily again, and at least stops the passage of larger amounts of air or liquids; second, the kind of food remnants present in the stomach at the time of perforation.

The *diagnostic* signs of perforative peritonitis, to the significance of which Ebstein[37] first called attention, are the following three: (1) Abdomen flat, rigidity of the abdominal muscles, which only relaxes at death. (2) Disappearance or diminution of liver dulness; this sign may be absent, however, if only liquid gastric contents and no air escape into the peritoneal cavity (Ebstein-Schotten[38]). (3) Vomiting. As is well known, vomiting, an ever-present symptom in perforative peritonitis, may be absent in perforation of the stomach (as shown by many observations), and that when it perforates into the free peritoneum. Previous vomiting may suddenly cease under these circumstances. But there are exceptions to this rule also (Schotten) if only very small amounts of gastric contents can escape through the perforation opening.

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[35] Gerhardt, *l.c.*

[36] Habershon, St. Barthol. Hospit. Rep., 1890.

[37] Ebstein, Zeitschrift für klinische Medicin, 1885, Bd. 9, P. 209.

[38] Ernst Schotten, Münchener medicinische Wochenschrift, 1893, No. 41.

3. *Perigastritis* may develop in cases of gastric ulcer in two forms, according to v. Mikulicz.[39] First, as loose adhesion between the neighbouring organs, whereby the stomach, losing a part of its mobility, is subjected to traction. Second, as tumour-like infiltrations, developing through the fact that the ulcer gradually advances in the direction of the abdominal wall.

As palpation fails in the first-named group, diagnosis is, as v. Mikulicz emphasises, very difficult. There is to be added to the fact, often found on operation, that adhesions exist which never caused stomach complaints. The demarcation against idiopathic cardialgias is hardly possible. Various writers have stated symptoms which are claimed to make the recognition of perigastritis possible or to facilitate the same. According to Rosenheim,[40] an extensive severe painfulness in the epigastrium, if the same extend to the right over the gastric borders, even if no dulness is present, points to an affection of the serosa of the anterior stomach wall; a large and irregularly shaped dulness in the region of the spleen is found, where formation of exudate has developed on the left side; simple adhesions manifest themselves through a severe pain felt in the depth, if the lower costal arch is pulled to the side and upward. Besides these symptoms, Rosenheim emphasises chiefly two signs which speak considerably for the presence of perigastritic adhesions: (1) If in a case of absolutely sure gastric ulcer pain continues sometimes after a very carefully applied treatment. (2) If improvement of the state of health is not effected through mechanical treatment of the stomach in cases where the same was indicated on account of a motor disturbance. According to Merklen,[41] in cases of perigastritis, pain follows ingestion of food just as in cases of ulcer. While, however, pain in ulcer is in connection with the quality of the ingested substances, it is claimed that in perigastritic adhesions the same is independent of it, for the pain is not occasioned through the irritative quality of the food, but through the tractions caused by the weight in the stomach.

Finally, Westphalen and W. Fick,[42] in consideration of two successfully operated cases, called attention to the exacerbation of pain by mechanical causes.

In spite of the above-named symptoms, the importance of which must not be underrated, we still have to content ourselves with a certain "probability" diagnosis, and the only fact that, as Lauenstein[43] states, other palpable changes cannot be demonstrated, will lead the diagnostician anew to the probability of adhesions. Certainty is given by the explorative laparotomy which, in such cases, is an acknowledged and necessary measure for the recognition of the condition and for its cure.

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[39] v. Mikulicz, Mittheilungen aus den Grenzgebieten, 1897, Bd. 2, S. 200.

[40] Rosenheim, Deutsche medicinische Wochenschrift, 1895, No. 3, S. 48.

[41] Merklen, Société médic. des hôpitaux, Janvier, 1899.

[42] Westphalen and Fick, Deutsche medicinische Wochenschrift, 1899, No. 52.

The conditions in the second type, the tumour-like adhesions, are more favourable. In recent time, Hofmeister[44] gave the following characteristic symptom-complex: "Along with more or less severe gastric disturbances, existing for years, there develops quite gradually a slowly growing tumour in the left epigastric region. After these disturbances have lasted for years, very severe pains situated in the region of the tumour are confronted, which, occurring particularly after ingestion of food, show the picture of colicky attacks. Vomiting was observed in all cases, hæmatemesis in one case. Finally, the nutrition suffers in a high degree, and grave emaciation is shown."

Relying on this symptom-complex, other writers (Ali Krogius,[45] Klaussner,[46] Brenner,[47] Lindner and Kuttner,[48] Schwarz,[49] B. Hirschfeld,[50] and others) have diagnosticated the affection with more or less probability, and in a few cases a successful operation was performed.

(4) *Ulcus Carcinomatosum.*—In about five or six per cent. of the cases of simple ulcer, *carcinoma*[51] develops, beginning at the edges of the ulcer. Rosenheim[52] emphasised that carcinomata, originating in an ulcer, are associated with a pronounced superacidity and gives the following diagnostic sentences:—

If undoubted cancer symptoms (tumour, progressing emaciation) are present, the presence of free hydrochloric acid at the acme of digestion, or superacidity, speaks for the development of this malignant neoplasm from an ulcer. On the other hand, absence of free hydrochloric acid, with undoubted cancer symptoms, does not exclude the origin of the cancer from an ulcer. Only the second sentence is correct. Lindner and Kuttner[53] observed, in some cases of circumscribed carcinomatous nodules associated with intact mucosa, normal secretion of HCl to the end, although the development from a previous ulcer could be excluded. Riegel[54] also reports a similar observation. My own experience has likewise shown, as will be more fully discussed in the Chapter on Cancer of the Stomach, that in a rather great number of cases of cancer of the stomach secretion of HCl persists till death, though no signs of a gastric ulcer or any other gastric affection have preceded the suffering from cancer.

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[43] Lauenstein, Archiv for klinische Chirurgie, 1893, Bd. 45, S. 221.

[44] Hofmeister, Beiträge zur klin. Chirurgie, 1895, Bd. 15, S. 356.

[45] Ali Krogius, Centralblatt für Chirurgie, 1896, S. 538.

[46] Klaussner, Münchener medicinische Wochenschrift, 1896, S. 863.

[47] Brenner, Wiener medicinische Wochenschrift, 1896, No. 48.

[48] Lindner and Kuttner, Die Chirurgie des Magens, Berlin, 1898, S. 173 and 214.

[49] Schwarz, Mittheilungen aus den Grenzgebieten, 1900, Bd. 5, S. 848.

[50] B. Hirschfeld, *ibid.*, 1900, Bd. 6, S. 462.

[51] Hauser, Das runde Magengeschwür, Leipzig, 1883.

[52] Rosenheim, Zeitschrift für klinische Medicin, 1890, Bd. 17, S. 116. (There the bibliography.)

[53] Lindner-Kuttner, Die Chirurgie des Magens, Berlin, 1898.

There is, therefore, no possibility to differentiate between ulcus carcinomatosum and an idiopathic carcinoma, from the point of examination of gastric contents. Ulcus carcinomatosum can be diagnosed only when a simple ulcer, if possible, accompanied by hæmatemesis, had been observed years before.

As in all respects the symptoms of ulcus carcinomatosum coincide with those of gastric cancer, they will be discussed in the chapter devoted to this disease.

5. *Hour-glass Stomach.*—The deformity of the stomach called hour-glass stomach (Fig. 57), is in some cases congenital and may be found incidentally at post-mortem; in other cases it has been acquired. In the majority of cases it is an ulcer situated before the antrum pylori or at the fundus, which is apt to cause an hour-glass constriction of this kind. Moreover, duodenal ulcers with cicatricial constrictions and subsequent stenosis may lead to pyloric insufficiency, by which a kind of hour-glass form of the stomach or duodenum is produced. Such cases have been described by Reiche.[55] I convinced myself of a similar condition at an operation of a duodenal stenosis situated low down, a condition which considerably embarrassed the surgeon in the recognition of the status. Carcinomata of the stomach fundus or pyloric section—a case of this kind observed by Riegel[56] —as well as adhesions between the stomach and neighbouring organs, compression from without and within (corset-constricted stomach) may cause hour-glass stomach.

The diagnosis of hour-glass stomach is difficult, but by no means impossible, as shown by several recent instances (Bourget[57] Schmidt-Monnard,[58] Bouveret,[59] Jaworski,[60] Watson Cheyne[61]). The subjective symptoms are not unlike those of pyloric stenosis; its type may be perhaps recognised from the previous history (ulcer, corrosion, etc.). Of greater importance are the objective symptoms. In some cases the characteristic hour-glass form may be seen on inspection, or better on inflation of the stomach. Various authors regard the fact as diagnostically important that on lavage of the stomach, after the same has been apparently completely emptied, suddenly large amounts of gastric contents make their appearance.

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[54] Riegel, Die Erkrankungen des Magens, S. 690.

[55] Reiche, Jahrbuch der Hamburger Staats-Krankenanstalten, 1900, S. 180.

[56] Riegel, Die Erkrankungen des Magens, Wien, 1897, S. 797.

[57] Bourget, quoted by Hemmeter, Diseases of the Stomach, Third Edition, P. 643.

[58] Schmidt-Monnard, Münchener medicinische Wochenschrift, 1893, No. 19.

[59] Bouveret, Lyon médic., February 2, 1896.

[60] Jaworski, Wiener medicinische Presse, 1897, No. 51.

[61] Quoted by Hemmeter, *l.c.*, P. 643.

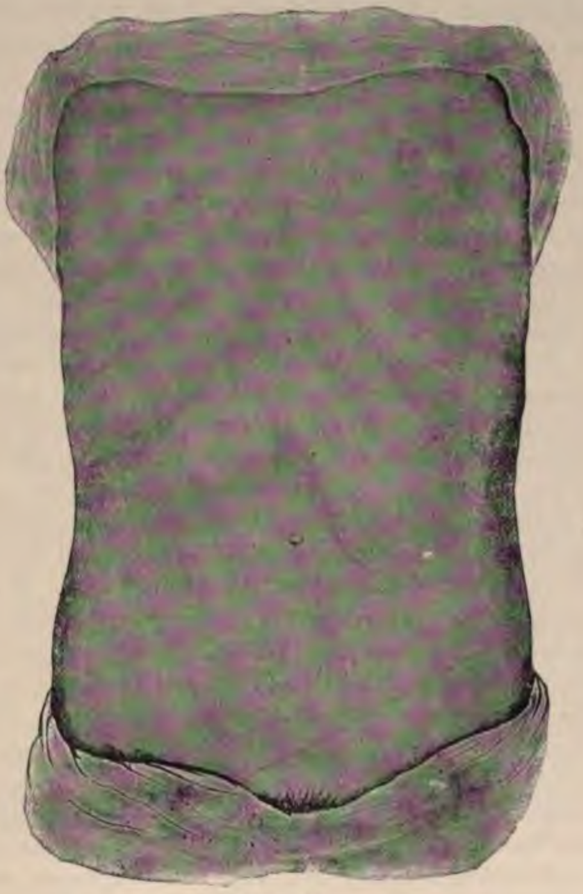

Fig. 57.

As Bouveret correctly states, this is but a subordinate symptom, as the same condition is found occasionally in perigastritis if it involves the stomach, as well as in simple dilatation of the stomach. This author considers inflation of the stomach as the decisive diagnostic means. While the succussion sound is heard precisely as in every case of pronounced gastrectasy deep below the navel, the inflated area in a bilocular stomach is situated exclusively high above the navel in the left hypochondrium, under the false ribs. The same condition exists on filling the stomach with water. Furthermore, the inflated area is very small in comparison to that in pyloric stenosis, and finally the seat of constriction is not, as in pyloric stenosis, on the right, but in or even to the left of the median line.

Jaworski[62] has applied to advantage gastro-diaphany for recognition of the hour-glass stomach. Ewald,[63] likewise, lately recommended inflating first and then to transilluminate the stomach, whereby the constricted pyloric section can be distinguished to the right from the red visible area of the upper gastric section.

6. *Subphrenic Abscess.*—v. Leyden[64] deserves the great merit of having first, in 1880, called attention to a clinical picture characterised by the following symptoms: the disease follows perforative peritonitis, or escape of pus from the intestines into the peritoneum. A purulent exudate forms in the lower part of the right or left thoracic cavity under symptoms of inflammation (pain, fever), but without cough or expectoration. On examination full percussion sound in the lower right or left thoracic part to the costal border, dulness in the corresponding posterior lower region; absence of vesicular murmur and fremitus; metallic sound can be made out if one percusses and auscultates simultaneously; succussion sound is distinct. The lung is distinctly intact above these parts, expands and descends on deep inspiration. The respiratory murmur is vesicular and the fremitus maintained down to the fourth or fifth rib; from here on the respiratory murmur suddenly ceases. The dulness which corresponds to the exudate changes with various positions of the body. The signs of equally distributed pressure in the pleura are wanting or only slightly pronounced. The movements of the corresponding half of the thorax are not co-ordinate, the intercostal spaces are almost obliterated, and the heart is but slightly pushed to the other side. If the exudate is on the right side, the liver projects far down into the abdomen, and can be felt at, or below, the umbilicus. In the course of the disease, the exudate may perforate into the respiratory passages, and, causing sudden and copious expectoration of fœtid pus, will make the diagnosis, if still doubtful, an absolute certainty. Since v. Leyden's publication frequent observations of subphrenic abscesses have been made. Maydl,[65] in a monograph on this subject, collected, up to 1894,

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[62] Jaworski, Wiener medicinische Presse, December 19, 1897, S. 1601.

[63] Ewald, 13. internationaler medicinischer Kongress, 1900; Berliner klinische Wochenschrift, 1900, No. 33, S. 739.

[64] v. Leyden, Zeitschrift für klinische Medicin, 1880, Bd. I, S. 320.

Maydl, Ueber subphrenische Abscesse, Wien, 1894. (There the corresponding literature.)

one hundred and seventy-nine cases of subphrenic pus collections, described in the literature. It is true, among them, all subphrenic abscesses of various origin are mentioned, while practically only those subphrenic abscesses which showed the clinical picture of pyo-pneumothorax should have been taken into account.

The most frequent ætiological factor (about 20 per cent.) of the said cases of subphrenic abscess was *perforation* of a *perforated* gastric or duodenal ulcer; in a few cases cancer of the stomach and œsophagus.

The above-mentioned symptom-complex, associated with most careful consideration of previous history and physical examination, is so characteristic that the diagnosis has been made in many cases and surgical intervention has been undertaken successfully.

Treatment.

Therapeutics in treatment of gastric ulcer has to fulfill three tasks; first, a prophylactic one; then one extending to the treatment of the ulcer itself; finally, one in regard to the predisposition to the formation of the ulcer.

Prophylaxis rests upon a mild and non-irritating diet (milk is most suitable), if signs of ulcer or symptoms of follicular ulceration manifest themselves; sudden deviation in the temperature of food (hot soups, ice) must be avoided, and, if necessary, regular evacuations must be effected by dietetic measures. If anæmia is present, a careful attempt to administer iron preparations may be permitted; on an average, however, I observed that such preparations are not well borne in gastric ulcer; iron mineral water proved to me the most effective one of these. If the dyspeptic symptoms do not cease under this treatment, it will coincide with the regular treatment for ulcer, to be described below. The latter varies according to presence or absence of an evident hæmorrhage. We will consider the first possibility more in detail, because occasionally a human life depends on the most painstaking consideration of the factors in question. The treatment may be suitably divided into four stages:—

First Stage (Hæmorrhage.)

The most important factor during the stage of hæmorrhage is absolute rest in bed and immobility of the stomach (patient must not move and not even arise for urination or defæcation). The next important factor is *absolute abstinence from food or drink.* Even ingestion of ice is rather injurious than beneficial; an absolutely empty condition of the stomach is the best remedy for contraction of the blood-vessels. Medicinal treatment ought to be omitted, whenever possible, during this stage. When the bleeding becomes abundant, a good ergot preparation may be administered.*

℞ Extracti secalis cornuti dialysati (Ph. German)	1.0
Aquæ destillatæ	5.0
Acidi carbolici	0.1

Sig.: A hypodermic syringeful to be injected into the stomach region.

In most recent time, gelatine, on account of its much-discussed, but by no means explained effect upon the aneurism of the aorta, has been administered by the mouth and hypodermically. (Poliakow,[66] Bauermeister,[67] Curschmann,[68] Wagner.[69]) The first mentioned writer reports a case of profuse gastric hæmorrhages which stopped within twenty-four hours after three administrations of 200 cubic centimetres each of a 10 per cent. gelatine solution, and when they reoccured, only two or three times during the succeeding four weeks, they readily yielded to the introduction of gelatine. Wagner reported 7 cases of severe hæmatemesis, in 4 of which bleeding immediately ceased after the first injection of gelatine. Further observations have to be looked for. A 5 to 10 per cent. solution, one tablespoonful every hour, has been recommended for the administration by mouth. For the hypodermic application the authors recommend a solution of 1 to 2 per cent. prepared with physiologic sodium chloride solution, which had been repeatedly sterilised (*cave* temperature above 100° C.), kept in a sterile flask, and heated to 38 C. directly before its use. For the injection either a Dieulafoy syringe, or a simple piston-syringe, or the regular apparatus for infusion of salt solution (glass funnel, tube, and cannula) is to be used. The pain of the puncture is, according to Wagner, very slight if ether spray is applied. Strictest antisepsis [and asepsis] is of course indispensable.

As the essential factor in the effect of gelatine has been referred to the lime contained in the gelatine, recent writers have recommended solutions of lime, especially calcium chloride, for internal hæmorrhages (A. G. Wright, Mayo Robson, Kehr).

Besides this, ice-bag and ice compresses on the stomach region gave good services. If we have to deal with otherwise strong persons, and if the pulse and general nutrition are good, no alimentation, not even by rectum, should be administered; only, if on account of profuse and rapidly repeated bleeding danger is present, nourishment may be given, but exclusively by rectum. The nutrient enema which I administer has the following ingredients:—

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* Whether ergot is of any special use in grave cases, seems to me very doubtful, as far as I have observed. [D. D. Stewart considers ergot as valueless in gastric hæmorrhage.—Medical News, January 23, 1892, "System of Practical Therapeutics," Vol. II, P. 949.]

[66] Poliakow, Medicin Obosren., 1898, Referat Klinische therap. Wochenschrift, 1888, S. 1809.

[67] Bauermeister, Deutsche medicinische Wochenschrift, 1899, Therapeutische Beilage, P. 84.

[68] Curschmann, Münchener medicinische Wochenschrift, 1899, No. 11 (referat).

[69] Wagner, Mitteilungen aus den Grenzgebieten der Medicin und Chirurgie, 1900, Bd. 6, Heft 4 and 5, P. 700.

250 grammes of milk.
2 yolks.
1 teaspoonful of salt.
1 tablespoonful of red wine.
1 tablespoonful of aleuronat flour.*

This mass, with an addition of 5 to 10 drops of opium tincture, is heated lukewarm and permitted to run slowly into the rectum two or three times a day, after a preceding cleansing enema of warm water.**

If the pain is severe, morphine hydrochlorate (0.01 to 0.03) or codeine hydrochlorate or phosphate (0.03 to 0.05) should be injected, or the latter may be administered in a suppository, perhaps in combination with extractum belladonnæ (0.03 pro dosi). As a rule, there is constipation, but usually purgatives are not desirable. If evacution seems to be necessary, irrigation of lukewarm soap-suds, glycerine, olive-oil, castor oil, with codliver oil and carbonate of soda*** is to be recommended.

Second Stage (Fourth to Seventh Day).

Patient remains in bed in a strictly recumbent position, a Priessnitz Umschlag being applied to the epigastrium. Feeding by the mouth may be resumed three days after the last occurrence of hæmorrhage. For the succeeding week the diet should be exclusively fluid, and always of the temperature of about 38° to 40° C. (100° to 104° F.). Most suitable food for this period is milk (infant's milk); if the same is not well borne, an addition of a small amount of tea may be allowed. Furthermore, beef-tea or puro may be permitted, as well as artificial or natural bouillon, perhaps with addition of meat-extract. Kemmerich's or Koch's peptone, plasmon, eulactol, [vigoral, Valentine meat-juice, mosquera beef-jelly], or similar preparations. Albumen and yolk of egg in emulsion may be allowed. In this stage v. Leube administers the meat-solution named after him. Cocoa, chocolate, and wine are better avoided, but the numerous acidulous waters, particularly those with a smaller amount of carbonic acid, such as Vichy,

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* [I frequently use tropon mixed with starch.]

** About other forms of nutrient enemas, see General Part, page 301.

*** Take two tablespoonfuls of castor-oil and two tablespoonfuls of codliver-oil, dissolve a piece of soda, the size of a bean, in 250 cubic centimetres of water, and pour carefully into the castor-codliver-oil mixture, stirring till a perfect emulsion has been effected.

Bilin, Neuenahr, Vals, Krondorf, Fachingen, Salvator, etc., may be recommended as very suitable.

Third Stage (Second and Third Weeks).

At this time a *typical cure for ulcer,* according to v. Leube[70] and v. Ziemssen,[71] should be instituted about in the following manner. Every morning and evening, patient in a recumbent position in bed, takes 250 cubic centimetres of (40° R. = 122° F.) Carlsbad Mühlbrunnen in which 5 to 10 grammes of natural or artificial Carlsbad salts have been dissolved. Among the less well-to-do patients, the latter alone dissolved in 250 cubic centimetres warm (40° R.) water is entirely satisfactory. Hot poultices are permanently applied, and so hot that the skin appears slightly scalded.* Occasionally, I make use, instead of the poultices, with equally good advantage, of *spongiopilin,* cut into any requisite shape and dipped into hot water, which is applied to the epigastrium and covered with oiled silk and flannel bandage, and renewed every three or four hours during day-time, and in the beginning even during night-time. The diet consists mainly of milk and the above-mentioned fluid substances; if there is great weakness, feeding by the rectum may be advisable and expedient.

If the tenderness in the epigastrium and the painful pressure and the cardialgias have ceased, the patient may be permitted to leave the bed and to rest on the sofa, even may be allowed to walk a little around the room. The Carlsbad cure is continued, if possible, for a period of at least four weeks. Milk should still be the prevailing fare, but there may be an addition of soft zwieback, cake [biscuit] crumbs, dipped rolls, meat preparations, such as calf's sweetbread or brain, dumplings made of finely divided meat, tropon, fish (perch, bluefish, trout), oysters, and clams in small quantities; light red wine diluted with one of the above-mentioned mineral waters may be allowed.

Fourth Stage (Fourth Week).

In the fourth week, if the subjective and objective conditions are good, there may be an addition of mashed potatoes (50 to 100 grammes), stewed apples, pears, prunes in purée form; vegetables that can be prepared in purée form (spinach, carrots, green peas, turnips, lentils, and beans) may be permitted; even a light farinaceous food may be added. Meat with tougher fibres may be gradually administered, but it must be tender and "rare." *For many years patients must avoid raw fruit, all kinds of strongly acid and spiced food and drink, greasy food, ice-cream, and all very cold and hot beverages.*

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* v. Leube applies hot poultices only when three months have passed since the last hæmorrhage. In shorter intervals between the hæmorrhages, he recommends the Priessnitz Umschlag; if intervals of only a week, ice-bag for the first days. As a rule I commence v. Leube's cure two weeks after the bleeding, and I never observed any unfavourable effect.

[70] v. Leube, v. Ziemssen's Handbuch der speciellen Pathologie und Therapie, VII, 2, S. 120; Mittheilungen aus den Grenzgebieten, etc., 1897, Bd. 2, S. 7.

[71] v. Ziemssen, Volkmann's Sammlung klin. Vorträge, No. 75.

What is the essential factor in v. Leube's treatment for ulcer? The views on this subject vary greatly. Rost[72] rejects, for instance, the hot poultices and replaces them through hydropathic compresses. F. Franke[73] observed better results in application of ice than with hot applications. In order to decide these differences, greater series of gastric ulcers would have to be treated by each of these methods. Experience, it is true, speaks in favour of the poultices, under which treatment within a few days a striking decrease of the epigastric and dorsal tenderness, as well as of the subjective painfulness, can be observed. On the other hand, in recent years I made thrice the observation that immediately after the application of hot poultices pronounced hæmatemesis developed in patients who had had no previous gastric hæmorrhage—the cases, however, were cured within the normal time. The administration of Carlsbad water and other alkalies is, according to my experience, only indicated in cases where constipation and hyperchlorhydria are present, and milk is not well borne. I could report numerous cases which healed smoothly within the usual four weeks without the introduction of any medicine or Carlsbad water. [It appears that the physicians in America are not very enthusiastic in the use of Carlsbad water in the treatment of gastric ulcer, especially as patients often become troubled with frequent movements of the bowels.]

As a general method in the treatment of ulcers without hæmorrhages, the above-described "rest-cure" is likewise best to be resorted to. In the majority of cases the result will be satisfactory; in a not small number, however, it was discouraging, or there are relapses soon after the return to the former method of life. In all these *refractory* cases of gastric ulcer, I advise, according to English writers (McCall Anderson,[74] Donkin[75]), a total abstinence cure of two to three weeks, during which the patients are in bed, are fed exclusively by rectal enemata (nutrient enema described above) three or four times a day.* Only if thirst is very tormenting, small pieces of ice or some Selters or another mineral water may be administered. It is remarkable that the desire for nourishment is satisfied through the enemata and that in many cases the general condition does not suffer. Part of the cure is the application of hot poultices or spongiopilin. Not earlier than the tenth day there may be a cautious and slow return to feeding of fluids by the mouth (milk, tea, bouillon, claret). After a few days, however, we may resort to more compact food. In a great number of gastric ulcers of this kind, after several typical rest-cures had been undergone, recovery occurred after this abstinence cure, though there were occasional recurrences, which, however, responded very rapidly to treatment.

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* According to Rost (Berl. klin. Wochenschrift, 1899, No. 31), the maximum of resorption is the highest when three nutritive enemas are given in a day.

[72] Rost, Berl. klin. Wochenschr., 1899, No. 31.

[73] Franke, v. Volkmann's Sammlung klin. Vorträge, 1900, No. 270.

[74] McCall Anderson, British Medical Journal, May 10, 1890.

[75] H. B. Donkin, The Lancet, September 27, 1890.

As a paradigma of the method in cases treated by me, the following may be mentioned:—

Case 1.—Mrs. R. O., suffered for fifteen years from stomach troubles. The complaints consist of drawing pains occurring on an empty stomach as well as after meals. Exacerbation of pair after ingestion of solid food. Six years ago, hæmatemesis. The general condition has suffered so much in the course of years that patient is hardly able to attend to her occupation. On examination, gastroptosis and myasthenia were found. In the epigastric region there is localised pain about three fingers' breadth above the navel, slightly on the right of the median line. Lungs, heart, genitals normal. Treatment: abstinence cure of ten days, nutrient enema three times a day; nothing except water by the mouth; no medicines. There is neither thirst nor hunger; general condition on the tenth day, excellent. From this day feeding by the mouth; milk for two days, then zwieback; from the fourth day, meat. Patient has been entirely well for the last two and one-half years, excepting quickly passing relapse, and has gained 9 kilogrammes in weight. Patient eats all kinds of food with impunity. The painfulness, mentioned above, has entirely disappeared.

Case 2.—Miss N., 30 years of age, cook. For the last six years, symptoms of ulcer, consisting of severe pain and vomiting after ingestion of fluids, as well as on empty stomach. Patient is considerably emaciated, not capable of working. Physical examination exhibits intense painfulness five centimetres below the ensiform process, exactly in the median line. Even 2.5 kilogrammes, by means of the algesimeter, produces severe pain; posteriorly on the left, at the level of the twelfth thoracic vertebra, likewise great painfulness. First, v. Leube's treatment for ulcer, without effect; then treatment by means of nitrate of silver, equally inefficient. May 1, 1893, abstinence cure. Patient receives only Vichy water by the mouth for two weeks; nutrient enema three times a day. From May 15th, soups, soft-boiled eggs, zwieback; later meat, light vegetables, mashed potatoes, etc., allowed. With the exception of one day (after scrambled eggs), absolutely painless. The algesimetric scale increases during the treatment from 2.5 to 6.5 kilogrammes. Patient discharged on May 29th.

The said abstinence cure, in favor of which Ratjen[76] and Rost,[77] of Ewald's ward at the Augusta Hospital, have spoken, yields, in my experience, the best results under careful clinical observation. In private practice, however, the proceeding is not without danger. Several times I observed grave attacks of syncope, which demanded an energetic intervention and immediate stopping of the rectal feeding. We must provide at least a trained nurse in the private practice.

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[76] Ratjen, Deutsche medicinische Wochenschrift, 1896, No. 52.

[77] Rost, Berl. klin. Wochenschrift, 1899, No. 30 and 31.

The other forms of artificial feeding, particularly those administered subcutaneously (oil, solution of sugar), have not yet been naturalised in the treatment of gastric ulcers.

In lighter cases of gastric ulcer or of follicular ulcers, or finally, in patients who, for extraneous reason, cannot undergo a typical ulcer cure, systematic treatment with a solution of nitrate of silver, as particularly recommended by Gerhardt,[78] is frequently an efficient procedure, according to my observations in many cases of this kind. I begin with a solution of 0.25 to 120.0, 1 tablespoonful in a wineglassful of distilled water three times a day, and that exclusively on an empty stomach. After this the dose is increased to 0.3 to 120.0; two bottles are taken, and finally the dose is increased to 0.4 to 120.0; one or two bottles are taken, according to necessity. I saw even desperate cases of ulcer being promptly cured under this treatment, with which, of course, a suitable diet (mainly milk, fresh-water fish, purées of potatoes and vegetables) has been used. It is true, a slight disadvantage of the remedy is the bad taste and consequent nausea, which is, however, readily neutralised by the addition of a few drops of peppermint.* [Silver nitrate may be administered in some other way, namely by the stomach tube. The tube, if carefully and cautiously introduced into the stomach afflicted with ulcer, does not bring about special danger, even if a hæmorrhage should exist at the time of introduction. In some cases at that time, ice-water or hot water has been introduced to check the bleeding. D. D. Stewart [79] never hesitated to use the stomach tube in chronic gastric ulcer, for the purpose of washing the stomach with solution of silver nitrate (1 : 1000), or solution of alumnol with succeeding introduction of bismuth. I have used the same method in many cases, always with benefit, never with harm, and I recommend the same very urgently, provided the physician can properly introduce the stomach tube.]

Very frequently in the beginning of the treatment with silver, diarrhœa is observed, which, however, never becomes very severe, but spontaneously ceases, in the majority of cases, after further administration.

In order to convince myself of the efficiency of the silver nitrate treatment I took in several cases systematic algesimetric measures in the epigastrium and the back, and I could ascertain the tenderness diminishing from week to week. In the curve below (Fig. 58), which is that of a patient with pronounced gastric ulcer, this gradual decrease of anterior and posterior tenderness has been graphically described.

The curve shows how the tenderness commencing with 3.5 kilogrammes gradually diminishes to slide, after a treatment of four weeks, to 6 kilogrammes, at which niveau it stands for some time, to descend after slight variations to 7 kilogrammes. The dorsal algetic points show a greater contrast in falling already after treatment of ten days precipitately down to the normal, *i.e.*, above 10 kilogrammes.

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* For removal of the metallic taste of the silver, rinsing the mouth with weak salt solution after each dose may be recommended.

[78] Gerhardt, *l.c.*

[79] Stewart, Sajous's Practical Cyclopædia of Medicine, 1906, Vol. 6. P. 42 and 44. (F. A. Davis Co., Philadelphia.)

Among other drugs, *bismuth preparations* and anodynes are to be considered. I prefer bismuth carbonate to bismuth subnitrate, because the former inhibits the intestinal function to a lesser degree.

Bismuth, having been considered of old as a specific in ulcer and its sequelæ, has recently been recommended anew by Kussmaul and Fleiner,[80] in large doses, as an excellent remedy in irritated conditions of the stomach and gastric ulcer. The method consists of administering to the patient, after previous lavage, 10 to 20 grains of bismuth subnitrate suspended in 200 cubic centimetres of lukewarm water, by means of the stomach tube. The tube is compressed, and, after remaining in the stomach for a few minutes, is removed after the clear water has been syphoned out. If there is no objection to leaving the water in the stomach, or if the tube is not well

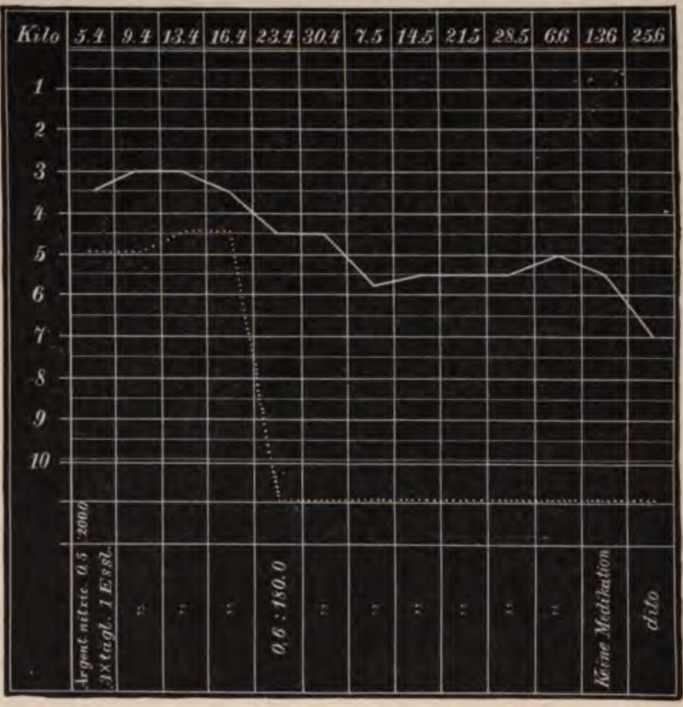

Fig. 58.

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[80] Fleiner, Verhandlungen des 12. Kongresses für innere Medicin, 1893.

borne, the latter may be removed immediately. The bismuth is administered first every day, later every other day, or every third day, and continued until the disappearance of the irritative manifestations. According to Fleiner, the administration of bismuth is indicated in the following cases:—

1. In all idiopathic, sensory, motor, and secretory irritative conditions of the stomach (gastric ulcers of long standing, ulcerating carcinoma, hæmorrhagic erosions).

2. As a specific remedy in all gastric and duodenal ulcers.

3. Bismuth (10.0 grammes in a glass of water), taken on arising, on an empty stomach, is recommended in cases of gastric ulcer and duodenal ulcer, in which the tube is contra-indicated and gastric hæmorrhages are present.

The bismuth treatment is *contra-indicated* in gastric affections accompanied by diminished secretion of HCl, with the exception of hæmorrhogic erosions and ulcerating tumours.

M. Mathes[81] has confirmed the favourable reports of Fleiner, and bases the same upon experiments showing that the introduced bismuth incites secretion of mucus, and that the mixture of bismuth and mucus furnishes a protecting cover over existing abrasions. Rosenheim,[82] Savelieff,[83] Crämer,[84] Witthauer,[85] Stintzing,[86] Riegel,[87] join in this view. Penzoldt[88] does not express himself either for or against the method. Olivetti[89] observed improvements during and after treatment with bismuth, which, however, did not last. Bourget[90] finds fault with the constipating effect of bismuth and doubts the possibility of a protecting effect in ulcers of the lesser curvature. My own observations, too, are not so favourable as those of the first-named authors. I had, in my practice, not a few failures, and could not always confirm the favourable tolerance of the remedy by the stomach as emphasised by Fleiner. In one case I observed severe diarrhœa, in two other cases insufferable gastric pressure. v. Leube also has not obtained any remarkable results from the bismuth treatment. The majority of the writers prefer the direct drinking of the bismuth solution to the introduction by means of the tube.

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[81] Matthes, Centralblatt für klinische Medicin, 1894, No. 1.

[82] Rosenheim, Pathologie und Therapie der Krankheiten des Speiserohre und des Magens, 1896, 2 Auflage, S. 362.

[83] Savelieff, Therapeutische Monatshefte, October, 1894.

[84] Crämer, Münchener medicinische Wochenschrift, 1896, No. 25.

[85] Witthauer, Therapeutische Monatshefte, October, 1896.

[86] Stintzing, Jenaer Dissertation, Fischer, 1893. (Quoted after Penzoldt.)

[87] Riegel, Die Erkrankungen des Magens, 1897, S. 726.

[88] Penzoldt, Penzoldt-Stintzing's Handbuch der Therapie Innerer Krankheiten, 2 Auflage, Bd. 3, S. 324.

[89] Olivetti, Therapeutische Monatshefte, April, 1898.

[90] Bourget, Therapeutische Monatshefte, 1900, No. 7.

[After the administration of many hundred doses of bismuth subnitrate (15 to 20 grammes and more) in the Philadelphia Polyclinic, as well as in private practice, I must conclude that, while small doses of bismuth subnitrate have an inhibitory effect upon intestinal peristalsis, large doses do not act in the same manner; on the contrary the large dose has rather a beneficial effect upon the peristalsis. This fact is not only proved by the regular daily evacuation of the fæces, but can also be readily observed by the application of Röntgen rays.* H. A. Hare, in an editorial in the Therapeutic Gazette several years ago, objects principally to the use of large doses of bismuth in the treatment for gastric ulcer from the theoretical standpoint, that when a small dose of bismuth acts constipating, a large dose must do so still more. Practical experience disproves this theoretic conclusion. In some cases of constipation from other causes I made use of the experience obtained in the treatment of gastric ulcer, and administered a teaspoonful of bismuth subnitrate three times a day before meals with rather encouraging effect. I turned the attention of the late Dr. D. D. Stewart to this fact and he adopted my observation in the treatment of many cases.]

The treatment with *iron preparations* in gastric ulcer has not many adherents. Among them are te Gempt,[91] Ewald,[92] Penzoldt.[93] From my own experience I cannot report very favourably on the use of iron preparations. Many of the ulcer patients in my practice had to atone for the previous use of iron preparations with exacerbation of pain, before they came under my observation. Even when I administer iron, after healing of the ulcer, for the improvement of the cholorotic condition, I almost exclusively use Blaud's pills—I very seldom saw any special result.

Recently Bourget[94] recommended lavage with a 2 per cent. solution of chloride of iron** for the treatment of gastric ulcer in the stage of hæmorrhage; but as he simultaneously administers alkalies in larger doses, which neutralise the hydrochloric acid, it is very difficult to judge the effect of the former.

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* Dr. G. E. Pfahler, who had great experience in this regard, in a personal communication to me, stated that he has never found that the evacuation of the fæces has been made slower than in normal conditions.

** As well known, Gerhardt recommended ferri-chloride solution in cases of gastric ulcer associated with anæmia.

[91] te Gempt, Berliner klinische Wochenschrift, 1886, No. 15.

[92] Ewald, Eulenburg's Realencyclopädie, 3 Auflage, Bd. 15, S. 321.

[93] Penzoldt, Penzoldt-Stintzing's Handbuch der Therapie, 2 Auflage, Bd. 4, S. 321.

[94] Bourget, Therapeutische Monatshefte, 1900, No. 7.

Among the anodynes, morphine, given hypodermically, is the most prompt and surest remedy against severe gastralgia. Leubuscher and Schäfer,[95] as well as Hitzig,[96] proved, quite independently from one another, that morphine, particularly given hypodermically, has a peculiarity of considerably diminishing the HCl secretion, so that on account of the great frequency of increased secretion of HCl in cases of ulcer, morphine would be indicated even from this point of view.

Essentially different results were observed by Kleine[97] and Riegel.[98] The former observed in a dog with the usual gastric fistula that morphine exhibited rather a stimulating effect upon the secretion of HCl, than an inhibiting one. Riegel's experiments on dogs with Pawlow's fistula showed that morphine administered hypodermically or in suppository, almost always considerably increases secretion. Riegel[99] found similar, though not quite as pronounced, effects on examinations in the human being. The conclusion is that administration of morphine for any length of time is not indicated in cases of gastric ulcer, but rather that it be replaced by belladonna preparations or atropine, which have been ascertained by numerous experiments on animals and human beings to have an inhibitory effect upon secretion.

[It may be mentioned here, that in quite a number of cases bromides given by the mouth increased the secretion of hydrochloric acid in the stomach-contents, whereas when bromides were given by the rectum, the secretion was never increased, but frequently decreased. Boardman Reed made the same observation.]

In less severe cases chloroform water (1.0 to 120.0), 1 tablespoonful three times a day, may be given as an anodyne.

Stepp[100] reports favourable results with a combination of chloroform water and bismuth:—

℞ Chloroformii	1.0
Aquæ destillatæ	150.0
Bismuthi subnitrici	3.0

M. D. S. One or two tablespoonfuls every hour.

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[95] Leubuscher and Schäfer, Deutsche medicinische Wochenschrift, 1892, No. 46.

[96] E. Hitzig, Berliner klinische Wochenschrift, 1892, No. 49.

[97] Kleine, Deutsche medicinische Wochenschrift, 1897, No. 21.

[98] Riegel, Die Therapie der Gegenwart, 1900, S. 337; Zeitschrift für klinische Medicin, 1900, Bd. 40, S. 347.

[99] Riegel, Verhandlungen des 17 Kongresses für innere Medicin, 1899; Zeitschrift für klinische Medicin, 1899, Bd. 37, S. 5 and 6.

[100] Stepp, Verhandlungen der Nürnberger Naturforscherversammlung, 1893; Therapeutische Monatshefte, November, 1893.

Carlsbad, as a watering place, has many adherents. It is best to select among the thermal springs those with a medium temperature (Mühlbrunnen, 27.8° C.; Schlossbrunnen, 36.9° C.; Markbrunnen, 50.0°C.; Kaiserbrunnen, 49.7° C.; Elisabethbrunnen, 42° C.), but by no means the hottest ones.

Three or four glasses, with additional Sprudel salt (the powdered salt, Sal Carolinum pulverisatum) are taken in a day. It ought to be stated, however, that Carlsbad is not at all adapted for peracute cases of gastric ulcers. Patients of this kind should be in the bed, not in the baths! But I frequently saw favourable results for the ulcer convalescent from Carlsbad as a health resort. But Carlsbad is contra-indicated in cases of stenosis of the pylorus after ulcer, and likewise if carcinomatous degeneration of the ulcer edges is suspected. Besides Carlsbad, Vichy and Neuenhar may come into consideration. [Bedford and Saratoga—waters heated.]

Frequently the cold chalybeate waters of Franzensbad and Elster are recommended, particularly in anæmia and irritative symptoms of the stomach, as well as in the convalescence after ulcer. The patient, as a rule, drinks in Franzensbad the Franzensquelle and a little of the Salzquelle (either one heated slightly); in Elster, the Moritzquelle, Königsquelle, Marienquelle, Albertquelle, likewise with some Salzquelle. The iron springs of Pyrmont, Cadowa, Driburg, Reinerz, Liebenstein, Spaa, Steben, Schwalbach, Bocklet, etc., as well as the arsenious waters of Roncegno and Levico, are frequently used. I know, however, of numerous unfavourable effects of iron and arsenious waters, and counsel, therefore, great caution. The corresponding mineral water baths (thermal, iron, mud or "moor" baths), in connection with the drinking of the waters, are often a serviceable adjuvant.

When is an ulcer considered as cured? The absence of cardialgia, even after ingestion of solid food, is a very valuable subjective symptom in this respect. But of greater importance is the absence of painfulness in the epigastric area, which previously had been painful. In order to find a safe measure in this matter, I consider it necessary to mark the painful area upon the skin (best by a silver nitrate pencil), and to test from week to week, perhaps by means of the algesimeter described by me. An ulcer may be considered cured, if the tenderness on pressure amounts to at least 5 kilos. The dorsal tenderness has to be ascertained also, and the algesimeter must show at least 10 kilos.

Medicinal Treatment of Complications of Gastric Ulcer.

To these belong, first of all, repeated hæmorrhages, difficult to control, with succeeding collapse and perforation of the ulcer. In life-threatening hæmorrhages Ewald recommends ice irrigation of the stomach; simultaneously the bandaging of the limbs with rubber bandages or elastic rubber tubes is indicated, just as in cases of other internal bleeding. We have already mentioned the subcutaneous application of gelatine (page 454). In grave conditions from exhaustion after severe hæmorrhages, the hypodermic or intravenous infusion of common salt solution (3/4 per cent.) is a suitable and frequently efficient remedy.

In private practice, the hypodermic infusion is to be preferred to the intravenous infusion, on account of the easier technique and the independence from special apparatus. In hospital practice the intravenous injection is probably the better proceeding, as it more directly stimulates the heart action. On an average, measures of this kind seldom become necessary, as bleedings from ulcers only exceptionally reach such a dangerous degree.

If a perforation has occurred, the first condition is absolute rest and subsequent quiescence of the gastro-enteric action, arresting peristalsis by large doses of opium (extracti opii 0.03 to 0.05 in suppository every two hours or oftener) or hypodermic injection of morphine. Ice-bag to the abdomen,. stimulants hypodermically and by the rectum. I recommend oleum camphoratum (1 to 10) or the double salts of a caffeine. For instance:—

℞ Coffeini natrio-salicylici or natrio-benzoici........	3.0 to 5.0
Aquæ destillatæ	20.0

M. Sig.: One or two Pravaz syringefuls to inject hypodermically.

Rectal feeding, with addition of warm port wine, is to be administered as described heretofore (p. 455).

[In perigastritic adhesions hypodermic injections of thiosinamine are worth a trial.*]

Surgical Treatment of Gastric Ulcer and Its Complications.

Before discussing surgical intervention in gastric ulcers, the preliminary question as to the results of internal treatment must be answered. The statistic records show, as they frequently do, but an incomplete picture of the curative figure, as the patients vary, the definition of cure can be stretched, the diagnosis is not always certain, and finally, the methods of treatment differ very much. The following figures are therefore of value only with a certain reserve:—

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* [Bernheim, Pennsylvania Medical Journal, February, 1906; Literature.]

	Cure	Death	From
Welsh	85%	15%	6.5% perforation peritonitis. 3.5% hæmorrhage 4 to 5% pylephlebitis and other complications.
Débove and Rémond	50%	50%	13% perforation peritonitis. 5% hæmorrhage. 5% inanition. 20% tuberculosis. 7% other complicatiens.
v. Leube	74%	2.4%	
Ewald	75%	?	

In this compilation the enormous death-rate of Débove and Rémond is astonishing; no doubt it must be referred to particularly unfavourable material or other uncontrollable influences. The figures of Welsh, v. Leube, and Ewald, seem, according to my observation, to come very near to reality. The conclusion is that, according to figures, the indication for operative intervention is not very frequent, even if we assume, and as has been repeatedly emphasised by surgeons, that the cures are not lasting, because in such cases repeated ulcer cures may be yet accompanied by success. Surgical intervention for the remaining cases of incurable gastric ulcer must be gratefully welcomed as a marked progress.

If we first consider the uncomplicated gastric ulcer, about the following maxims are decisive for surgical intervention:—

1. *Hæmorrhages.*—We have to distinguish (*a*) profuse single and repeated hæmorrhages; (*b*) small and frequently repeated bleedings. In the former case the indication for surgical operation is difficult to define, as v. Leube[101] correctly emphasises; for, according to general experience, it is usually to be awaited if the bleeding does not spontaneously cease. Whether, if such is not the case, the surgeon always or even but frequently will succeed in checking the hæmorrhage, is very questionable, and as far as is known to me, even prominent surgeons decline interference in severe acute hæmorrhages. Only a few clinicians and surgeons, among whom are Dieulafoy[102] and Petersen,[103] favour surgical intervention, *i.e.*, circumsuture of the bleeding and contiguous portion, or gastro-enterostomy. The demand of surgical treatment is much more obvious and evident in frequently repeated small hæmorrhages, through which patients fall into a state of anæmia of a high degree. Based on this indication successful

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[101] v. Leube, Mitteilungen aus den Grenzgebieten, 1897, Bd. 2, P. 8.

[102] Dieulafoy, Clinique médicale de l'Hôtel Dieu, 1897-1898; Bullet. de l'Académie de Médicine, 1898, and Presse médicale, 1898, No. 6.

[103] Petersen, Deutsche medicinische Wochenschrift, 1899, No. 24-25. (Heidelberger Chirurgische Klinik.)

operations have frequently been performed by various surgeons, such as Küster, Doyen, Carle, v. Mikulicz, Guinard, Bond, Roux, and Tuffier.[104]

As a rule, gastro-enterostomy will be satisfactory in such cases. My own experience includes four cases, three of which were operated on because of persistent and frequently recurrent bleedings, the fourth one on account of severe pain and vomiting. Complete cure took place in three cases, after gastro-enterostomy; in the fourth there was essential improvement. In general, however, the indication is not often given. Operation during and immediately after hæmatemesis may be not opportune, and if patients have overcome the attack, they believe—sometimes prognosis shows them to be right—that this attack was the last to occur. Usually, operation, therefore, will be limited to those cases in which repeated attacks of hæmatemesis and melæna cause a threatening loss of strength.

But there is often great difficulty in diagnosis, as we mentioned above. The differentiation between bleeding from an ulcer and bleeding from passive congestion, originating in cirrhosis of the liver or thrombosis of the portal vein, is not always easy, and there is therefore evident danger in performing an entirely useless operation, which of itself is not always without risk of life to the patient. The precondition for surgical intervention is, accordingly, that the diagnosis of an ulcer or small erosions is made certain as much as possible. [Compare Rodman, "Gastric Hæmorrhage and Gastric Ulcer: Non-perforating Hæmorrhage."[105]]

2. *Pain and Vomiting.*—In this respect also clinicians and surgeons agree essentially, the restriction having been accepted on both sides that operation may be advised only after the armamentary of internal treatment has been exhausted. I would add another restriction, namely, that the diagnosis of ulcer should be based to a certain extent upon objective symptoms also, particularly upon typical hæmatemesis and melæna. Generally, this indication, too, will not often give cause for operation, as, according to experience in cases of uncomplicated gastric ulcer, a single or repeated ulcer cure leads to success, *i.e.*, disappearance of pain and vomiting.

Much depends on the social standing of patients, who frequently belong to the poorer classes. Every practitioner knows that such patients, even if willing, cannot impose upon themselves a long-continued general and local forbearance, with corresponding diet, which we demand as an urgent precondition for the cure of an ulcer. In such cases the very existence of the patients is at stake, and therefore the surgical treatment becomes for them a very command of self-preservation. On the other hand, sufficient observations have not been made whether the complaints of the patients are removed forever through surgical intervention, *i.e.*, gastro-enterostomy, for it is only this one with which we would have to deal. The results of Doyen,[106] Lauenstein,[107] Stendel-Czerny,[108] to which I can add an encouraging observation of my own, indeed incite favourable expectations.

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[104] Tuffier, quoted from Terrier and Hartmann, Chirurgie de l'estomac, Paris, 1899, P. 207.

[105] Rodman, Philadelphia Medical Journal, June, 1900.

The surgical treatment of complications extends, first, to cicatrised pyloric stenosis, pylorospasm, development of carcinoma from an ulcer; second, to perforation and peritonitis, perigastritis, and finally, the subphrenic abscess. The discussion of the first three complications is here omitted, as they will be discussed in other chapters. We discuss first:—

(1) *Surgical Treatment of Perforation* (*Perforative Peritonitis ex Ulcere Rotundo.*)—The indication for surgical intervention is given by the experience that the perforation of a round ulcer into the abdominal cavity, if treated internally, yields an unfavourable prognosis, exceptions being exceedingly rare. Exceptions occur under the following circumstances: (1) When the stomach was completely free of food at the moment of perforation. (2) When the peritonitis becomes localized for reasons not yet explained. As, however, the first-named possibility can be very seldom ascertained, as, for instance, in a case of Pariser,[109] and the second is manifested only from its course, in almost all cases in which perforation is certain, a quick co-operation of the clinician and the surgeon is urgently required. For the peril, as the convincing statistics of Weir and Foot,[110] as well as particularly the statistical report of Terrier and Hartmann, based on a material of 169 cases, demonstrate, increases with the time which has elapsed between perforation and operation. While, for instance, only 30 per cent. of 53 cases perished, which were operated less than twelve hours after perforation, the death-rate grows to 58 per cent. after waiting twelve to twenty-four hours, and to 76 per cent. after twenty-four to forty-eight hours. After a duration of forty-eight hours, the rate of death diminishes again, manifestly because then we have to deal with less severe cases or such as become circumscribed in the meanwhile, which naturally have a more favourable prognosis.*[111]

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* [Martin Tinker,[111] in an elaborate paper on "Perforating Gastric Ulcer and Its Surgical Treatment," gives the following figures of mortality: In 23 operations before 1896 (Keen and Tinker), there was a mortality of 39.13 per cent.; in 131 operations after 1896 (Keen and Tinker), a mortality of 35.71 per cent., whereas Weir and Foot had a mortality of 71.51 per cent. in 78 operations.]

[106] Doyen, Traitement chirurgical des affections de l'estomac, 1895.

[107] Lauenstein, Deutsche Zeitschrift für Chirurgie, 1896, Bd. 44.

[108] Stendel, Beitr. zur klin. Chirurgie, Bd. 23, Nos. 1 and 2.

[109] Pariser, Deutsche medicinische Wochenschrift.

[110] Weir and Foot, Medical News, Philadelphia, 1896, vol. i.

[111] [Martin Tinker, Inaug.-Dissert., Berlin, July 21, 1899; Philadelphia Medical Journal, February 3, 1900, PP. 251-261.]

While, accordingly, the operation must not be delayed too long, on the other side it should not be precipitated, and especially it should not be performed in the first shock. The best time is probably within the first ten or twelve hours, because shock will have passed in the meantime. If exceptionally, however, it lasts longer, the operation will have to be ventured even under these circumstances, though with less good prospects.

In cases where abscesses, as a complication of perforation of gastric or duodenal ulcers, have developed in the neighbourhood of the stomach, especially on the lower surface of the diaphragm (pyopneumothorax subphrenicus, v. Leyden), a cure can hardly be secured in any other way than by means of the knife.

(2) *Perigastritis.*—As we have mentioned above, adhesions occur in two forms, either as stringlike or lamelliform adhesions with pancreas, spleen, liver, gut, or anterior abdominal wall, or in form of firm and immovable tumours. Only the latter form can be diagnosed under favourable conditions; the former can only be suspected.

The indication for surgical intervention can, therefore, extend only to the cases with palpable tumour. About fifteen operations have been performed, based on this indication. There are a number of assured cures among these operated cases; in other cases they were without success; in still others the time passed since operation is too short.[112]

In the other group, a precise indication is much more difficult. It is derived more from the subjective complaints, from the inefficiency of the applied treatment and the increasing nutritive disturbances. General rules for surgical intervention cannot be set up; the condition of the case and the extent of the complaints are the deciding factor. Favourable reports on severance of adhesions have been made by v. Hacker,[113] Hahn,[114] Lauenstein,[115] v. Mickulicz,[116] Collins Warren,[117] and others. In lighter cases the so-called gastrolysis, *i.e.*, to sever adhesions, will be satisfactory; in severe cases, gastro-enterostomy seems to be the most expedient procedure. (Westphalen and Fick.) In one of my cases, in which continuous and severe gastric pain existed quite independently from the ingestion of food, the affection developed in conjunction with a grave trauma, gastro-enterostomy (Professor E. Hahn) achieved a complete cure.

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[112] Compare Schwarz, Mittheilungen a. d. Grenzgebieten, Bd. 5, S. 821 *et seq.*, and Hirshfeld, *ibid.*, Bd. 6, S. 462.

[113] v. Hacker, Wien. klin. Wochenschrift, 1887, Bd. 45, S. 141.

[114] Hahn, lecture delivered before the Free Association of Surgeons, Berlin, May 25, 1894.

[115] Lauenstein, Archiv für klinische Chirurgie, Bd. 45, S. 141.

[116] v. Mikulicz, Mittheilungen aus den Grenzgebieten, Bd. 2, S. 184.

[117] Collins Warren, Boston Medical and Surgical Journal, 1896, vol. 2, P. 308; quoted from Terrier and Hartmann, Chirurgie de l'estomac, 1899, P. 537.

[Among the surgeons best known in this line of work, we have to mention the brothers William J. and Charles H. Mayo, Rochester, Minn. Their experience is extensive and their success great.[118]]

(3) *Hour-glass Stomach.*—The surgical treatment of hour-glass stomach is a product of recent time.[119] The first successful operation, as far as I know, was performed by Schmidt-Monnard.[120] Since that time several favourable reports have been made by Wölfler, Hochenegg, Langenbuch, Lauenstein, Hofmeister, Jaboulay, Chaput, Roux, Schwarz, Watson, Catellani, Marion and Tuffier, and v. Eiselberg.[121] About twenty-five cases have been reported to the present time.

For the treatment of hour-glass stomach, the following methods have been recommended:—

1. Gastroplasty, a method which, similar to that of pyloroplasty, is performed so that on the site of the constriction a longitudinal incision is made through the anterior wall and it is then united transversely.

2. Gastro-anastomosis, a connection between the two bags, similar to that in gastro-enterostomy.

3. Gastro-enterostomy. The gained experience, particularly that of v. Eiselsberg,[122] who performed seven operations, speaks in favour of gastro-anastomosis and gastro-enterostomy.

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[118] [William J. Mayo, The Association of Surgical Lesions in the Upper Abdomen, Journal of the American Medical Association, June 11, 1904.]

[119] Compare Die Geschichte des Sanduhrmagens by Wölfler. Beiträge zur klin. Chirurgie, Vol. 13, S. 221.

[120] Schmidt-Monnard, München medicinische Wochenschrift, 1893.

[121] Quoted after Guillemont, L'estomac biloculaire, Paris, 1899. (The bibliography until and including 1899.)

[122] v. Eiselsberg, Archiv für klin. Chirurgie, 1899, Bd. 59, S. 825.

Appendix.

Hæmorrhagic Erosions of the Stomach.

Relying upon the regular finding of fragments of the gastric mucosa on lavage of the stomach, Einhorn[123] was the first to present the clinical picture of the hæmorrhagic erosions. In the essential points, C. Pariser[124] and E. Quintard[125] agreed with Einhorn.

The symptom-complex, according to Einhorn's descriptions, consists of pain after meals, emaciation, feeling of weakness. Of importance is only the distress after meals, for the other two symptoms are evidently only the result of the diminished nourishment on account of the fear of pain (sitophobia). How ambiguous, however, pain may be (Einhorn describes its intensity as slight, but Pariser as considerable), in the course of digestion, need not be said.

There is another factor, namely, the observations made by Einhorn and Quintard, that the secretory conditions, in spite of the conformity otherwise, do not correspond. There remains, therefore, only the consistent presence of eroded fragments of the mucosa. Indeed, this consistency is greatly astonishing, and this reason is sufficient to call attention to Einhorn's communications. Whether we will succeed in unsheathing a typical clinical picture, seems to me questionable. The newly-reported cases of Sansoni[126] exhibit various deviations from the descriptions given by Einhorn. Unfortunately, there is no post-mortem examination in any of the reports. At this time, therefore, it is, in my opinion, not yet allowable to establish a precise clinical picture; but only the fact may be registered, that there are conditions associated with more or less severe gastric complaints which are accompanied consistently by the presence of eroded areas of the gastric mucosa.

As to treatment, Einhorn and Quintard applied the silver nitrate spray (1:2000); Pariser, lavage with weak silver nitrate solution; and Sansoni, bismuth. Whereas Einhorn does not seem to consider a special diet of importance, Pariser emphasises the same particularly. In Sansoni's cases, too, a definite diet was prescribed.

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[123] Einhorn, Berliner klinische Wochenschrift, 1895, Nos. 20 and 21; Archiv für Verdauungskrankheiten, 1899, Bd. 5, No. 1.

[124] Pariser, Medicin Revue, 1897, No. 1; Berliner klinische Wochenschrift, 1900, No. 43.

[125] Quintard, New York Medical Record, September 15, 1900.

[126] Sansoni, Archiv für Verdauungskrankheiten, 1900, Bd. 6, No. 1.

CHAPTER XVIII.

Motor Insufficiency of the Stomach.

Conception and Nature.—The capacity of the stomach varies in the widest limits. It is hardly possible to tell when it is still normal, or when it begins to become pathologic. Even if a stomach surpasses the extreme limits of capacity (about 3 litres) it may still do justice to the demands made upon it. Liebermeister[1] reports the case of a coachman who could drink exceedingly large quantities of beer and could empty the largest tankard in one draught. The coachman was killed in an accident, and on post-mortem it was found that his stomach held six litres but was entirely normal otherwise. During his life there were never any symptoms of digestive disturbances. These conditions, which may be observed occasionally intra vitam,[2] and no doubt occur more frequently than has heretofore been supposed, are suitably designated as megalogastria (Ewald) or as "physiologic large stomach." A criterion for pathologic conditions, therefore, cannot rest upon the varying degrees of capacity of the stomach *alone.*

Quite different is it as to the judgment in regard to the motor function. It is, with a few negligible exceptions, a constant gauge, almost unchangeable from the first to the last breath. The healthy stomach is free from food in the morning, the healthy stomach empties a fixed small meal in about two to four hours, a fixed large meal in about seven hours. In this functional constancy there is therefore the most important measure as to the judgment of a morbid deviation. These deviations vary very much according to their degree. The stomach is still capable of freeing itself entirely from small and pulpy or fluid substances, but can only partially expel larger or solid masses; retention follows. But even in the first-named case, the time required for the digestion of the meal is longer than under normal conditions. We speak, according to the example of O. Rosenbach, of motor insufficiency of the stomach, and the delayed but complete expulsion of the stomach-contents is designated as relative insufficiency; the incomplete expulsion which leads to the formation of retention, as absolute insufficiency. In similar manner I suggested the designation of the two types of insufficiency as the first and second degree. Corresponding with the latter designation is the term "stasis insufficiency," suggested by Naunyn. We shall apply either term in one and the same sense.

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[1] Liebermeister, Die Krankheiten der Unterleibsorgane, Leipzig, 1894, S. 70.

[2] F. Riegel, Deutsche medicinische Wochenschrift, 1894, No. 15.

The most varied changes as to capacity, shape, and position of the stomach may be connected with these disturbances of the gastric function. The stomach may be dilated in cases of severest insufficiency, but need not necessarily be so; it may be dislocated downward or upward, or may be in normal position; it may assume a vertical shape or an abnormal transverse dimension, or may keep its normal shape. But—and this is of fundamental importance—all the named changes in size and position may be present, though the course of gastric motor function, and, as we will add, the secretory function also, may be entirely normal.

The conclusion, therefore, is that the decisive factor in abnormal changes in size and position of the stomach is the dynamic function, the maintenance or the loss of its peculiar peristaltic power. Not that the changes in size and position are superfluous symptoms; they have and retain their clinical significance as adjutant factors, only they do not play, as formerly, the first rôle in the diagnostic ensemble—they have become *symptoms of secondary rank.*

Up to this point there exists at the present time a perfect unanimity among all eminent authorities since Kussmaul's pioneer work. The difficulties begin at the moment when an attempt is made to express the function and the size of the stomach under *one* term. Different authors, in order to preserve the term "dilatation of the stomach" for the clinic, have attempted this evidently impossible task, which, as could not otherwise be expected, failed.

I take from the numerous attempts of this kind only the following, in order to show the hopelessness of all present and future endeavour to force the function and size into one system:—

Kuttner[3] defines, in his otherwise excellent book, dilatation of the stomach as follows: "In dilatation of the stomach there exists a constant dilatation of the stomach cavity, demonstrable even if the stomach is empty, and an insufficient emptying of the organ, so that always, usually also in the fasting stomach, more or less considerable quantities of stagnant food-remnants are encountered. The exact diagnosis of the dilatation of the stomach is based solely upon the simultaneous proof of the dilatation and motor disturbance of the stomach."

This definition is entirely correct when the clinical symptoms of the ectasia ventriculi are always combined with the finding of the dilatation of the gastric cavity. How is it, however, when such is not the case? If the symptom-complex of dilatation of the stomach is present, the gastric cavity, however, is not dilated, or is even diminished? Then we have the same clinical picture as in ectasia, only with the difference that there dilatation, here normal capacity, or even diminution, prevails. It would be, of course, absurd to speak in such cases of ectasia. But how to call the clinical picture? The term of atony would not be suitable, either; for atony, as Kuttner also emphasises, means a condition of deficient dynamic function, in which the chyme is propelled, though slower, at least completely into the intestine.

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[3] Lindner and Kuttner, Die Chirurgie des Magens, Berlin, 1898.

The salient point in the whole discussion is, accordingly: is there a symptom-complex of gastric dilatation without change in the size of the organ? If this question is affirmed, as moreover Kuttner himself admits, and as it has been proven by Schreiber, Naunyn, and others, a long time ago, and as we have demonstrated on the base of repeated observations, the term "dilatation of the stomach" in the present meaning is not justified, or a new term must be created for those forms which, clinically, exhibit all the symptoms of gastric dilatation, but anatomically do not show any change in capacity.

We are furthermore induced, through a series of other points of view, to repudiate this term. First, the fact, well known to every trained physician, that adherence to the symptom of dilatation of gastric cavity has practically caused great confusion. Second, in reality we determine *intra vitam* usually only the position of the greater curvature, perhaps also the distance between the lesser and the greater curvatures: only in rare cases the capacity for fluids. The position of the greater curvature is determined in comparison with the level of the umbilicus: a very bad standard, as the position of the navel is a very varied one, and surely descends lower in the course of severe diseases, in which adipose tissue is lost. Finally, another point of view is that, as later will be discussed more circumstantially, the paramount majority of cases of grave motor disorders which were current formerly as dilatations of the stomach, turned out to be stenoses of the pylorus.

As we herewith approach more the ætiologic diagnosis, the term "stomach dilatation" becomes more superfluous than formerly, when there was an uncertainty as to the frequency of motor insufficiency, dependent upon stenosis and atony.

[Einhorn suggested the name "ischochymia," in analogy to ischuria. Ischochymia does not express the real condition, as the meaning of the Greek "ischo" is to suppress or check; while in the morbid picture of retention of chyme, the chyme or secretion of gastric juice is present; *i.e.*, has not been suppressed, but retained, kept back. In some cases where there is a suppression of gastric juice, one could rather speak of "ischochylia." Strümpell[4] makes a very precise distinction between ischuria and retention of urine in saying, "Very remarkable are some observations of hysteric *ischuria* . . . . and the bladder never contained more than a few drops of urine."]

The deduction from these reasons is that the emphasis of the disturbance of the motility in the symptom-complex means a distinct progress, because it turns the attention of the diagnostician to the real nature of the disease and guards him against overestimation of the capacity of the stomach. We shall, therefore, discard in the following the nomenclature of dilatation of the stomach or gastrectasia as a definitive clinical term, and speak of dilatation as of a secondary symptom. As the motor insufficiency of the first degree, in a great majority of the cases, though not always, is dependent upon relaxed conditions of the muscles of the stomach, the terms atony and myasthenia may retain their places.

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[4] [Strümpell, Lehrbuch der Speciellen Pathologie und Therapie der inneren Krankheiten, Leipzig, 1887, Bd. 2, Part I, P. 481. Translation, Appleton, New York, 1896, P. 305.]

It is evident that the clinical picture of gastric motor insufficiency does not depend only upon the mechanic disturbances which follow the same, but to no less an extent upon the nature of the causing affection.

The cause of the mechanic disturbances of the stomach may be of various natures: idiopathic affections of muscles and muscle-nerves, central paralysis, spastic or organic stenosis of the pylorus through cicatrices, tumours, corrosions, mechanic pressure or adhesions, abnormal course in the pyloro-duodenal angle or sharp-angled deviation in the duodenum itself, foreign bodies (gallstones, etc.), and many other abnormalities. We shall, in the separate chapters, make mention of the special affections causing gastric insufficiency.

I. Motor Insufficiency of the First Degree.

(Synonyms: Atony, Myasthenia of the Stomach.)

Motor-insufficiency may be acute or chronic. As acute disease, the grave type of gastric paralysis is better known than the light insufficiency. But it is undoubtedly the case that less dangerous forms occur frequently enough, though they mimic usually rather acute gastritis and are consequently recognised but seldom. In discussing acute gastric paralysis, we shall have to revert to it more in detail.

Ætiologically, it must be stated that motor insufficiency occurs as a chronic condition, either idiopathic, particularly following an irregular mode of life or a single or continued overloading with unsuitable food and especially drinks—besides alcohol, the abuse of milk, coffee, and soups must be emphasised—or represents a symptom or an accompaniment or sequela of the most varied acute and chronic diseases. To mention these separately is without special interest. It is frequently observed as a complication of diseases of the *alimentary canal.* Among the gastric affections I may mention, according to the frequency of their occurrence, descensus ventriculi (with or without dislocation of the right kidney), nervous dyspepsia, gastric ulcer, chronic gastritis. Of intestinal affections, I mention, in the order of their location, light pyloric stenosis, stenosis of the pars horizontalis inferior duodeni (in stenosis situated farther above stasis insufficiency usually follows very soon) or of the jejunum, enteroptosis, stenosis of the colon resulting from various causes, and intestinal catarrh of long standing. Habitual constipation also is, without doubt, apt to bring about gastric motor insufficiency. Furthermore, gastric adhesions may cause disorders of motility to a lesser or greater extent. Among the hepatic affections, I mention cholelithiasis (which is, according to Reichmann[5] and myself, accompanied very often with myasthenia of the stomach), then passive congestion in the area of the liver and its enlargement, passive congestion in the portal vein system. How far possible chronic inflammatory processes of the pancreas or spleen may be connected with gastric myasthenia is not known.

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[5] Reichmann, Berliner klinische Wochenschrift, 1897, No. 33.

It may be further stated that according to my observations muscular weakness of the stomach may be congenital, even may be present in all the members of a family.

Diagnosis.

First, it is to be emphasised that there are pronounced cases of myasthenia which may run their course *without any symptoms whatever.*

I examined repeatedly a woman, 25 years of age, who regards herself perfectly healthy, eats all kinds of food with impunity, and yet exhibits a pronounced gastroptosis, enlargement of dulness, marked succussion sound, and considerable delay of motor function (two hours after test-breakfast more than 150 cubic centimetres in the stomach). It is interesting, moreover, that her sister suffers from the same complaint, and has shown subjective gastric and nervous symptoms of a grave kind, and still shows them, though to a considerably smaller extent.

The latent state of the symptoms, however, is an exception.

In the characteristic cases there are among the subjective symptoms, sensation of oppression caused by the long-continued retention of food, or sensation of early satiety (fulness), frequent eructation, headache or dizziness, constipation. To the objective symptoms, which are the most important in this affection, belong the physical and chemical examination and its course.

(*a*) *Subjective Symptoms.*

In typical cases sensation of pressure is associated with the ingestion; when stomach is empty, there is a relative feeling of comfort. The greater the demands made upon the myasthenic organ, the smaller the strength to comply with, the more distressing the subjective manifestation. Simultaneously there exists the precipitate *sensation of satiety,* which compels the patient, in spite of his good appetite, to cease eating before he has taken the usual amount of food. In recent cases the pressure does not last very long (about half an hour to an hour); in more advanced cases, it may distress the patient for hours, from one meal to the other.

A comparatively frequent symptom is increased *belching,* either simple eructation of air or taste of the previously eaten food. The time after which eructation follows the previous meal is a pretty fair criterion for the delayed peristalsis. In some cases, especially if acid secretion is increased, regurgitation or even vomiting of the ingesta may take place; in other cases pyrosis is a very distressing complaint. After repeated eructations the sensation of pressure may diminish or cease entirely.

A frequent but by no means constant symptom is *constipation,* which occurs either as a sequela of the delayed gastric peristalsis, or appears as a concomitant symptom of gastro-enteric atony. Generally, however, the constipation is never of such a degree as in severe cases of gastric insufficiency.

In pronounced cases of gastric atony, pressure in the head, which may be associated with dizziness and transitory loss of consciousness has occasionally been observed (*vertigo stomacal*). This so-called *gastric vertigo* was claimed first by Boerhave, particularly emphasised by Trousseau,[6] and later by v. Leube and others as a symptom of chronic gastritis; other authors in recent time agree with Trousseau. I pointed out (p. 413) that the cases of gastric and intestinal vertigo, which I observed, were connected always with gastric and intestinal myasthenia. That gastric and intestinal atony is the most frequent substratum for establishment of auto-intoxication has been emphasised by Al. Pick,[7] supported by many observations, and by Julius Friedenwald.[8] As opportune observations of gastro-enteric vertigo are not frequently reported, I refer to the following two cases:—

CASE I.—Fr. K., school-teacher, 34 years old. Patient comes from healthy family, but mother suffered from gastric catarrh, so-called, for many years. Patient was perfectly healthy till 1876. At that time, without special causes, there appeared gradually gastric disturbances, which consisted of feeling of fulness after meals, bulimia when stomach was empty, tremor and weakness when the same was not satisfied, dizziness and constipation. After use of Carlsbad cure, improvement of several years' duration, till about 1882. The complaints commenced anew; patient used Carlsbad water again, with favourable effect, but only of short duration. For the last two years considerable exacerbation of the complaints, especially of the headache and dizziness. Dizziness makes its appearance early after breakfast, lasts for several hours, disappears for a short time after luncheon, only to reappear with greater severity after dinner. Patient had frequently to interrupt the school sessions. The sensation of dizziness often continued till late at night. Physical examination shows that, with the exception of the stomach, all other organs (nervous system was somewhat irritated) were unimpaired. The stomach is not tender on pressure, but very much relaxed. Greater curvature visible hand's breath below the umbilicus; up to this level, splashing sound. When the stomach is filled with fluids its fundus descends two or three fingers' breadth farther downward. The examination of the gastric-contents, repeatedly made, shows an empty stomach in the morning, large residuum one hour after every test-breakfast. Always abundant free HCl (Congo 1.5 per cent. HCl). Urine abundant, without sugar or albumen.

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[6] Trousseau, Gaz. des hôpitaux, 1862; Schmidt's Jahrbucher, Bd. 117, S. 34, and Med. Klinik des Hôtel Dieu in Paris, 1868, Bd. 3. Translated by Cullmann u. P. Niemeyer.

[7] A. Pick, Wiener klin. Wochenschrift, 1892, No. 46 and 47.

[8] Julius Friedenwald, Medical News, December 23, 1892.

CASE II.—M. K., official, 33 years. Father was suffering from gastric affection, mother healthy. Patient himself was never seriously ill (except children's diseases), but already at an early stage of his life complained of a "weak stomach," so that he had to avoid heavy food or overloading. Patient had to take medicines for constipation. Since February, 1892, the complaints of the patient are considerably exacerbated, as he states, after drinking cold beer. A sensation of pressure and fulness takes place, particularly after the main meal. Especially distressing to him was the *sensation of dizziness and flickering before his eyes. Patient became ataxic. For some time patient has noticed a peculiar uncertainty in his legs.* Simultaneously with dizziness there is heart-palpitation, congestion towards the head. Patient says he frequently feels creeping or formication in his feet, which are frequently cold, often becoming numb. Appetite is fair, bowels constipated, hæmorrhoids. Formerly much belching, but not so of late; flatus only on defæcation; patient lost a few pounds in weight.

On physical examination I found that pupils react promptly to light and accommodation. Patellar reflexes are retarded, but maintained; Romberg's symptom absent. Everywhere sense of touch and temperature good, no diminution of strength. Abdomen sunk in, around the umbilicus slight distension. No tenderness on pressure. After introduction of 200 cubic centimetres of water into the empty stomach, pronounced succussion and splashing, hand's breadth below the navel; splash-sound on change of position. Greater curvature three fingers' breadth below the level of the umbilicus; the upper limit of the splashing, about five fingers' breadth above the navel. Rectal examination shows moderately large hæmorrhoidal nodules. Fasting stomach empty; one hour after test-breakfast, large amount of gastric contents, more than 200 cubic centimetres, reaction acid, weak HCl reaction. Amount of HCl, 0.08 per cent. Repeated examinations of stomach-contents yield likewise low amounts of HCl.

It is worth while to mention that not seldom *asthma dyspepticum* occurs in persons with atony; I have reported several cases of this kind.[9]

(b) *Objective Symptoms.*

Physical examination can give us the information about size and position as well as about the elastic condition of the muscles of the stomach. We mentioned above that the capacity as well as the position of the stomach is physiologically variable. But changes in size and position may call attention to a possibility of a disturbance of the gastric dynamic function. Of greater importance is the examination for the elastic condition of the muscles of the stomach. Experience teaches that a myasthenic stomach shows less resistance to the stretching effect of water or air than a normal one, full of vigour. Such is, however, not always the case. If the muscles of the stomach, as well as the superlying membranes, are enlarged by the inflammation and very much thickened, such a stomach may appear entirely normal in regard to the stretching effect of water and air, though the specific function of the gastric muscles may nevertheless be impaired.

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[9] Boas, Archiv für Verdauungskrankheiten, 1896, Bd. 2, P. 444.

Methods of examination: inspection, palpation, auscultation, percussion, inflation with air and carbonic acid, tube palpation, transillumination.

Occasionally, if the abdominal walls are relaxed, the borders of the stomach, particularly the greater curvature, become visible (inspection from the head end of the patient). Sometimes a descensus ventriculi can be diagnosed on mere inspection; but in general, inspection offers only unimportant diagnostic points.

A trained physician may palpate the stomach, filled with air, and may differentiate its borders from those of the neighbouring organs, particularly from the transverse colon, which, usually being full, offers greater resistance.

A frequently applied trick is the production of *splash sound* (clapotement).

But as there are frequently wrong conceptions about the estimation of splash sound, a short discussion may not be superfluous. The splash sound is of diagnostic importance and points to a pathologic dilatation if it can be distinctly produced to a larger extent, namely, from the epigastrium downward to the symphysis, through short tappings with the tips of the fingers, and if, furthermore, on change of position the sound of a shaken half-full cask can be produced. The diagnosis becomes certain when the same manifestations can be made to appear if the stomach of the examined patient is fasting. But the splash-sound labouriously produced and not combined with the succussion sound is without diagnostic importance. Splash sound occurring even to a larger extent may be found along with succussion sound at the acme of digestion, though the gastric function is perfectly normal, particularly if descensus or vertical position of the stomach and thin abdominal walls are present; but in such cases it will be absent on an empty stomach.

The *auscultation* is usually restricted to the confirmation of the just-mentioned succussion sound while the patient makes active motions and changes his position.

Percussion must be exclusively finger percussion and is only to be done slightly. The most important factors in the percussion are the establishment of the greater, and, if possible, also of the lesser curvature and the ascertainment of the length and breadth of the stomach (Pacanowski[10]). One percusses from the region above the symphysis upward till a dull tympanitic zone is reached, which is marked by means of a dermographic pencil; then one goes farther upwards in an attempt to determine the lesser curvature, which attempt, of course, on account of the anatomical position of the stomach, succeeds only when the same has more or less descended, a very frequent occurrence, as is well known, in cases of atony.

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[10] Pacanowski, Deutsches Archiv für klinische Medicin, 1887, Bd. 40, P. 342.

The determination of the upper (gastro-pulmonary) and of the right (gastro-hepatic) border of the stomach has but an inferior practical importance. If distinct splash sound is audible and the lesser curvature determinable, we will easily succeed in determining, by percussion and palpation, the "stomach length" and "stomach breadth," *i.e.*, the greatest vertical and horizontal distances. The stomach length under normal conditions amounts, according to Pacanowski's investigations, to 11 to 14 centimetres in men, to 10 centimetres in women; the stomach breadth to 21 centimetres in the former, and 18 centimetres in the latter. In cases of mechanic insufficiency of the stomach these limits may be surpassed to a greater or less extent.

We do not possess an absolutely sure and irrefutable method for testing the *elasticity* or *tonus* of the gastric walls. A simple and, under certain precautions, useful procedure for the test of the gastric elasticity is the method of Penzoldt or Dehio, mentioned in Chapter IV (page 91); this method may be combined with diaphanoscopy (see below), by introducing, during transillumination, precisely measured quantities of water into the stomach and observing the change of position of the greater curvature. If this descends considerably downward when burdened by 100 or 200 cubic centimetres of water, it would point to a gastric atony.

In cases where percussion does not give a definite explanation, the inflation with carbonic acid or air is an approved and expedient proceeding. The inflation must be omitted in all cases where ulcus ventriculi or new adhesions between the stomach and other intestinal sections is suspected, or finally where meteorism of a large degree is present. For the purpose of carbonic inflation, the patient is made to drink first a teaspoonful of tartaric acid in about 150 grammes of water, and afterwards sodium bicarbonate in the same quantity of water.

If the patient is used to the tube, distension by means of air is to be preferred to that by CO_2; but in such cases still greater caution is necessary than in those with CO_2 inflation. An ulcer existing for a brief time, or one lately healed, is a distinct contra-indication for the inflation with air. It is of practical importance not to distend the stomach ad maximum, as it comes to lie so close to the anterior abdominal wall that the outlines disappear.

In patients with thin abdominal walls, palpation by means of the tube through the abdominal walls is often successful, as I have demonstrated.[11] A former assistant of mine, Dr. Schmilinsky,[12] was able to palpate the tube in ninety-seven out of one hundred cases of the most varied gastric affections. It is true, such a favourable result is obtainable only after great practice. By this method we may ascertain the position of the greater curvature and the location of the pylorus in a manner corresponding physiologic conditions. (Compare Part I, page 99.)

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[11] Boas, Centralblatt für innere Medicin, 1896, No. 6.

[12] Schmilinsky, Archiv für Verdauungskrankheiten, 1896, Bd. 2, Heft 2.

If the patient is used to the introduction of the stomach tube, the *transillumination of the stomach is an expedient proceeding for the determination of the stomach borders* [General Part, page 115]; the greater curvature, particularly, is made conspicuous. By transposition of the gastrodiaphanic tube in the stomach a very good survey as to the size of the stomach can be had, and occasionally it may be elucidated whether the stomach is enlarged or has descended. According to Kuttner and J. Jacobsohn,[13] a dropped stomach is differentiated from an ectactic stomach through the fact that the former does not change its position on inspiration, while the latter, connected as it is with the diaphragm, exhibits a distinct inspiratory descending. As, however, Kuttner[14] more recently emphatically states that a physiologic and large stomach exhibits the same gastrodiaphanic picture as a pathologic and dilated stomach, and as, furthermore, respiratory transposition occurs in cases of gastroptosis, and finally, gastroptosis may be complicated by gastric insufficiency, only a reconnoitering and by no means decisive significance can be attributed to transillumination.

[As mentioned in the General Part (pages 125 and 126), the application of Röntgen rays is not to be neglected.]

In order to demonstrate the *dynamic function* of the stomach, the Riegel-Leube's test-meal is used. The normal stomach is empty seven or eight hours after the ingestion of this test-meal; the atonic stomach exhibits more or less food remnants. Very appropriate is also the method of Mathieu and Rémond (*vide* page 151). If more than 200 cubic centimeters of fluid has been retained, we may justly speak of a motor insufficiency of the first degree. For the same purpose, Strauss[15] recommended the administration of a tablespoonful of currants in the evening. The demonstration of currants in the fasting stomach in the morning points to motor insufficiency. The method did not come in great vogue with the practitioner, and, as the currant test is positive in many and varied gastric affections (ulcer, carcinoma, superacidity, it can be utilised diagnostically only with great caution. The "oil method," recommended by Klemperer (page 214), is well-founded scientifically, but practically of little service. Ewald's "salol test" (page 212) possesses many sources of error.

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[13] Kuttner and Jacobsohn, Berliner klinische Wochenschrift, 1893, No. 39.

[14] Lindner-Kuttner, Die Chirurgie des Magens, Berlin, 1898.

[15] Strauss, Zeitschrift für klinische Medicin, 1896, Bd. 29, P. 244.

Of importance also for the examination of motor insufficiency of the first degree is the examination of the stomach-contents after a "test-supper" which, according to my direction, consisting of two cups of tea, two rolls, and some cold meat, is taken at eight o'clock in the evening. If the fasting stomach is examined the following morning, macroscopically visible food remnants are never found in myasthenia.

The *chemical examination* of the vomit or gastric contents appears to me of little importance in regard to the diagnosis of atony, because the chemism may vary according to the stage of the motor insufficiency. This is hardly astonishing if we contemplate that a mechanical irritation is exercised upon the gastric glands through the more or less lasting retention of the ingesta. These glands react at first by increased secretion, and then gradually, at the stage of fatigue, by diminished secretion. In the beginning of atony usually an increase of HCl is found. In other cases the acidity is normal, and may remain so for years. In a third class subacidity or even inacidity is encountered.

Some authors, more from a theoretical standpoint than from practical experience, have spoken of gastric fermentation even in this lighter form of motor insufficiency. I have to refute decidely this statement, as least as far as it pertains to the known forms of fermentation (gas fermentation, lactic and butyric acid fermentation). The fact that the well-known condition of the three strata of the stomach-contents, which is so frequently found in stasis insufficiency, is missing in these cases, corresponds to the absence of gastric fermentation. The microscopic examination shows either no bacilli, or only a few; sarcinæ and yeast are entirely absent, or are occasionally observed, if perhaps they have been introduced along with the food.

The presence of ferments is parallel with the production of hydrochloric acid. In cases of deficiency of HCl the enzymes (pepsinogen, labzymogen) may be abundant, or but slightly diminished; but they may be absent, too, and the consequence will be a condition clinically congruous with that of anadenia of the gastric mucosa or achylia gastrica, so that recognition of the primary cause becomes very difficult.

(*c*) *The Course.*

Myasthenia, as a rule, is a chronic affection; this accounts for the frequent variations of the symptom-complex, and that as a result of errors in diet, weakening influences, intercurrent diseases, etc.

Whether myasthenia of many years' standing and with unsuitable dietetics can turn to ectasia is not yet proven. We shall revert to this question in the Section on Motor Insufficiency of the Second Degree.

Even in the lighter cases of myasthenia the general health may become impaired. The body weight may occasionally suffer so much that the inexperienced may think of a grave consumptive disease. Very frequently, especially in men, the digestive disturbances are accompanied by nervous manifestations of various kinds, such as headache or congestions, deficient memory, heart palpitation, backache, in brief, the well-known picture of neurasthenia. In regard to cause and effect, there is no certainty; there is, however, no doubt that a diminution of gastric motor function may produce the clinical picture of neurasthenia, especially if there is a nervous disposition present (*vide* following section).

Differential Diagnosis.

As to the differential diagnosis, we have to turn our attention to megalogastria, nervous dyspepsia, chronic gastritis, and gastroptosis.

1. *Megalogastria* is distinguished from atony, first, through the absence of gastric disturbances; second, through demonstrating the normal course of the digestive function (normal expulsion of test-breakfast or test-dinner; empty stomach after test-supper).

2. The symptoms of *nervous dyspepsia* have so much in common with gastric motor insufficiency—besides, there frequently occur motor disturbances of the stomach in cases of pronounced nervous dyspepsia—that the diagnosis will be in doubt, and sometimes the diagnosis cannot be made at all with any degree of certainty. Following moments speak for nervous dyspepsia: changes in the symptoms, alternating good and bad days, or even weeks; independence of the complaints from quality and quantity of the ingested food; finally, the presence of other neurasthenic symptoms. Furthermore, of importance is the presence of certain algetic points in the sphere of the great sympathic abdominal plexus, plexus cœliacus or solaris, and the plexus hypogastricus; also dorsal, irregularly disseminated algetic points are frequent manifestations in nervous dyspepsia. In cases of uncomplicated atony these complaints do not occur. It must, however, not be forgotten that, as mentioned above, atony in a neurasthenic individual may produce nervous dyspepsia; what in such cases is primary and secondary, can only be answered on the most careful consideration of the case in question.

3. The differential determination *between atony and chronic gastritis* depends, on the one side, upon the physical examination; on the other side, upon the exploration of the stomach-contents. In idiopathic chronic gastritis—at least in the typical cases—the stomach is not enlarged, motor function is well preserved, occasionally even increased. In chronic gastritis the formation of mucus is frequently the prevailing factor, while it is absent in atony; but abnormal secretion of mucus may be also absent

in the former. Moreover, as gastritis may develop in cases of atony of long standing and of an advanced degree, and vice versa, the differentiation of either affection may meet with great difficulties.

4. The differential diagnosis between *atony* and *gastroptosis or enteroptosis* depends, on the one side, upon the examination of the motor function; on the other side, upon the determination of the borders and location of the stomach; it may be mentioned that gastroptosis and gastric atony often occur in combination with one another.

Treatment of Motor Insufficiency of the First Degree.

Prophylaxis.—The weak condition of the muscularis of the digestive organs may be congenital. Certain constitutional diseases (chlorosis, anæmia, pulmonary tuberculosis, cholelithiasis, diabetes, etc.) may be contributory to a development of myasthenia; furthermore, atony may be the result of exhausting hæmorrhages and infectious diseases, particularly typhoid fever, malaria, diphtheria, influenza; and frequent and rapidly consecutive childbirths may, by causing increase of space in the abdominal cavity, [occasionally] lead up not only to a general enteroptosis—this subject will be discussed circumstantially later—but also to relaxation of the muscles. The cause is frequently dependent upon incorrect method of life, defective teeth, insufficient mastication, hasty deglutition, abnormal burdening of the stomach with food and particularly beverages, koprotasis, and the consecutive abuse of purgatives. According to the type of the case, we may be able to inhibit the progress of the at first latent, or at least not pronounced affection, and before more serious functional disturbances have made their appearance, by carefully considering the ætiology, or if this is unknown and inconceivable, by a suitable regulation of diet and strengthening of the abdominal walls by tonic measures. The details of this principle coincide with the dietetic and other therapeutic measures, to be described immediately.

The proper treatment of a pronounced atony has to consider or to ascertain, above all, the ætiological factor, and whether the atony in question is a partial symptom of another disease, or whether it is idiopathic. The anamnesis, according to the paradigma given by me in Chapter III, page 53 *et seq.*, will give information in many cases.

To mention a few instances, in anæmia the suitable treatment of the same, according to the well-known principles, is a most important precondition for the removal of the atony. In women with gastroptosis, the exciting cause must be counteracted by strengthening the abdominal walls by proper bandages, by baths, etc.

When a definitive ætiological moment is missing, the digestive organs will usually, though not always correctly, form the point of start, as well as of attack for the treatment. The treatment proper includes diet, hydropathic and electric measures, massage, and medicines.

Patients with atony must eat frequently, but very little at a time. [Rest after each meal is often of great service.] It is expedient to recommend compact food, poor in water, and to restrict the quantity of fluids, whenever possible, to 1 or 1½ litres in twenty-four hours, including tea or coffee, soups, alcoholics, etc.; decrease or increase of the amount may be regulated according to the condition in the individual case. As in atony, if not progressed to the highest degree, thirst is not essentially increased, the majority of the patients learn to get along with the mentioned amount of fluid. But when in the individual case a greater desire for fluid nourishment exists and its refusal produces dryness in the mouth, an enema of water or nutrient enemata may be a suitable adjuvant. Rectal feeding of several days for the sake of disencumbering the stomach has been recommended by various writers (Schlesinger,[16] Ewald[17]).

Special consideration must be given to milk, which has been highly recommended by some for gastric atony, as it was for the majority of gastro-enteric diseases. In general, milk as a *"cure"* is contra-indicated in cases of atony on account of the weakened motor apparatus. In anæmia, post-typhoid atony of the stomach, in tuberculosis, a milk cure, if large quantities of milk are well borne—against all theories—may be indicated, especially as in such cases exceedingly favourable conditions are created for the increase of organic albumen in general and strengthening of the muscles in special. As a matter of course, cases of atony with fermentative processes in the alimentary canal, slight though they may be, must be excepted.

The selection of the special diet is dependent on the state of the gastro-enteric apparatus. The chemical report, if strengthened by repeated examinations of the gastric contents, may give valuable points for the diet. Special rules, however, cannot be deducted from them. It will be advantageous to progress experimentally, to give at first very small quantities, and to increase them as the complaints improve; likewise, the food must be prepared according to well-known principles (purées of vegetables, minced meat), and the normal food must gradually be approached. I allow fats (only butter, cocoanut oil, or palmin) in normal quantities, as there is absolutely no reason for their prohibition.

Likewise, I do not forbid alcohol on principle. I agree with Klemperer,[18] whose experiments have been supported by exact clinical observations, that small quantities of good alcohol (brandy, claret, beer) rather stimulate than inhibit the gastric motor function. Of course, I am speaking only of small doses (brandy, 15 to 20 cubic centimetres, of wine or beer, 200 to 300 cubic centimetres pro die).

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[16] Schlesinger, Wiener klinische Wochenschrift, 1895, No. 75.

[17] Ewald, Eulenburg's Realencyklopädie, 3 Auflage, Bd. 14.

[18] Klemperer, Zeitschrift für klinische Medicin, 1890, Bd. 17, Supplementheft.

The treatment of chronic (habitual) constipation, a very frequent symptom accompanying gastric atony, requires special attention when it is very obstinate and leads to disturbances (flatulency, colics, headache, dizziness, etc.). *Nothing is more wrong in such cases than the use of purgatives, which, unfortunately, is still so common.* Indeed, in almost all cases of habitual constipation caused by intestinal atony, dietetic measures will be quite sufficient.[19] [I have often found *small* salt-water enemata rather valuable.]

For the rare cases in which I do prescribe purgatives, the following seem to me most suitable: first of all the well-tried rheum (rhubarb), best as extractum rhei compositum; tamarind preserve, or essentia taramindinata (one whisky-glass 3 times a day); frangula preparation, especially cascara sagrada.

℞ Fluid extract cascaræ sagradæ,
Syrupi corticis aurantii............................aa 30.0
Sig.: One teaspoonful to one dessertspoonful three times a day.

or sagrada wine (1 tablespoonful 3 times a day); or, finally, podophyllin in pill form:—

℞ Podophyllini,
Extracti belladonnæ ....aa 0.3
Succi et pulveris liquiritiæ.............................. q. s.
u. f. pilulæ No. xxx.
Sig.: One to three pills three times a day, or in granules of 0.01.

Aloe, colocynth, jalap, scammonium, gamboge, bitter waters and other drastics are absolutely to be avoided in intestinal disturbances caused by atony. [Exodin has been of service to me, and deserves recommendation.]

An important part of our therapeutic endeavour consists of the strengthening and stimulation of the insufficiency of the gastro-intestinal muscularis. How far are we able to influence the same?

Contrary to many authors, I regard *lavage of the stomach for this purpose not only superfluous,* but injurious. As there is no stagnation, but the stomach empties itself completely, though after delay, I cannot conceive any reason for the need of lavage in such cases. Whether we can influence *the gastric muscularis as to a lasting and strengthening effect* by other procedures, such as intragastric douche (highly recommended by

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[19] Boas, Diagnostik und Therapie der Darmkrankheiten, Leipzig, 1899, P. 249 *et seq.;* compare also English translation (Basch), New York, 1901, 251 *et seq.*

Rosenheim), is still uncertain; the fact that it may act favourably, perhaps by suggestion, will not be disputed.

The hydropathic procedures consist, in their simplest form, of the well-known Priessnitz Umschläge, then of cold water frictions, of douches to the epigastrium, especially the "Scotch douche," [slapping with the wet towel], as well as of various forms of wet packs, as they are applied successfully in well-managed institutions. It would lead us too far to discuss the theory of the effect of cold water cures in intestinal affections; it may suffice to say that the same, applied correctly and methodically, form one of the most excellent measures for atony of the stomach, not only that associated with general neurasthenia, but also for the idiopathic one.

The *electric treatment* of the alimentary canal may be applied as extraventricular or intraventricular, and in cases of atony the faradic current may probably be preferred to the galvanic current. It is difficult to say whether the endogastric treatment, in favour of which Einhorn[20] recently spoke anew, deserves to be preferred to the simple faradization of the abdominal walls. It is a fact that we can obtain favourable results by means of extraneous faradisation of the abdominal walls which may best be combined with massage (see below). The main effect of faradisation seems to me to be the influence upon the intestinal peristalsis, which indirectly improves also the gastric peristalsis.

The purpose of *massage* of the alimentary canal is the better nourishment and strengthening of the muscles by an increase of the blood circulation, and to stimulate the peristalsis of the various sections. The influence upon the intestinal peristalsis, which improves occasionally in not too advanced cases (unfortunately, not lasting), by skilfully and correctly applied massage, is most evident.

In many cases—especially in atony associated with nutritive disturbances and nervous symptoms—the treatment in well-managed sanatoria has the quickest effect, because the patient, being taught to eat, gains rapidly in strength.

By medicinal treatment, we chiefly aim through the promotion of the gastro-enteric peristaltic function, to decrease the disturbances. Second, the purpose of the medicinal treatment is to consider the particularly pronounced symptoms. As to the former respect, the strychnine preparations have obtained a certain reputation. The best preparation is extractum strychni in doses of 0.01 to 0.03, either in pills or powders:—

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[20] Einhorn, Berliner klinische Wochenschrift, 1891, No. 23; Deutsche medicinische Wochenschrift, 1893, No. 33-35; Zeitschrift für klinische Medicin, 1893, Bd. 23, Heft 3 u. 4.

℞ Extracti strycnni	0.1
Extracti gentianæ	
Pulveris radicis althaeæ	q. s.

u. f. pilulæ No. xxx.

Sig.: One or two pills after meals three times a day.

or:—

℞ Extracti strychni	0.03 to 0.05
Bismuthi carbonici	0.5

M. f. pulv.

Sig.: One powder three times a day.

When superacidity is present, the extract of nux vomica may be combined suitably with antacid remedies, such as magnesia usta, sodium carbonate, or, better, sodium citrate. I like to prescribe:—

℞ Magnesia usta,	
Natrii citrici	aa 10.0
Extracti nucis vomicæ	0.1 to 0.3

M. f. pulvis.

Sig.: One teaspoonful half an hour after meals three times a day.

When severe pains after meals, burning or pressure are present, we may add to the said powder 0.3 to 0.5 of codeine hydrochlorate or phosphate; if fermentation shows itself to a large extent, bismuth salicylate (0.5 three times a day) or salicylic acid (1.0 to 2.0 pro die), or resorcin (0.5 three times a day) may be recommended.

Besides strychnine, orexinum tannicum (0.2) has been recommended by Penzoldt as stimulating the gastric motor function and as inciting the appetite; but not all writers agree as to the efficacy of orexin.

Another medicine, which is claimed by Klemperer[21] to be a motor stimulant and simultaneously an antizymotic, is creasote. Pick[22] has successfully used lavage and creasote in powder form (0.05 pro dosi) in many cases of gastric disturbances connected with chlorosis.

Likewise Pick saw favourable results from ichthyol in gastric atony with concomitant fermentative processes in the alimentary canal. He prescribes

℞ Ammonii sulpho-ichthyolici	0.1
ad capsulas gelatinosas.	

Sig.: One or two capsules three times daily.

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[21] Klemperer, *l.c.*

[22] Pick, Wiener klinische Wochenschrift, 1881, No. 50.

Motor Insufficiency of the Second Degree.

(Stasis Insufficiency; Insufficiency from Obstruction.)

We mean by motor insufficiency of the second degree, or stasis insufficiency, as already stated, a condition in which the stomach is not capable any longer of expelling its entire contents; food material has been retained in the stomach, which is subjected to abnormal decomposition. This abnormal retention, in connection with the pathologic decomposition of the ingesta, produces that symptom-complex which was formerly known as dilatation of the stomach. The cause for the pathologic insufficiency may be threefold: either the muscularis is impaired, or the pylorus is stenotic, or, finally, the stomach is paralysed through central influences. The latter cause is rather rare, and further on we will have to discuss the more severe forms of gastric paresis, which usually develops acutely. Of more practical importance are the two first-named forms. The muscularis may be impaired through atrophy, or fatty or colloid degeneration; furthermore, by penetration of carcinomatous masses, caustic effect of poisons introduced from without, etc. The question whether a simple atony through overburdening of the organ may turn into a real paralysis, an insufficiency of the second degree, is answered diversely. Considered purely from the theoretic standpoint, such a thing might be possible, and this development is accepted in facto by numerous eminent authors, among others Ewald, Riegel, Rosenheim, Wegele. Others, however, (Schreiber,[23] Stiller,[24] Ullmann,[25] P. Cohnheim,[26]) dispute this transition. As on account of evident reasons the pathologic-anatomic experience can not give any aid, the chief point will be in the clinical observation, which teaches that simple atonies very seldom reach a degree corresponding to the clinical picture of stasis insufficiency.

The pylorus may be constricted from within or without; from within most frequently by ulcers, which, by retraction, leave a scar; by spasms, which develop in connection with ulcers or erosions, or in hyperchlorhydria or hysterics (Schnitzler); by caustic actions through poisons; by thickening of the gastric walls, especially of the pyloric section (Linitis plastica, Brinton); furthermore, by tumours (carcinoma, polypus, myoma, etc.); finally, by foreign bodies (gall-stones, hair-tumours). From without the pylorus may be compressed or deviated by adhesive processes which start partly from the stomach and partly from neighbouring organs, by tumours originating in the pancreas, the liver, the gall-bladder, the intestines, the lymph-glands, the mesentery, etc.

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[23] Schreiber, Deutsches Archiv für klinische Medicin, 1877, Bd. 79.

[24] Stiller, Die nervösen Magenkrankheiten, Stuttgart, 1884.

[25] Ullmann, Münchener medicinische Wochenschrift, 1895, No. 15.

[26] P. Cohnheim, Archiv für Verdauungskrankheiten, 1899, Bd. 5, Heft 4.

We may, according to the duration, distinguish two forms of stasis insufficiency: an *acute* and a *chronic* form.

(*A*) *Acute Gastric Insufficiency.*

(Acute Gastric Paresis; Acute Dilatation of the Stomach.)

While up to a short time ago, the occurrence of acute gastric paresis was doubted, recently so many cases of this affection, established on post-mortem examinations, have been reported, that at the present time the clinical picture must be considered as certain. It is true, the opinions as to the causes of acute gastric paresis differ very much, and at the present time no decisive view on the ætiology of the cases in question can be decided upon, the less so as even the post-mortem findings show a varied picture. In former post-mortem examinations not enough attention has been paid to the mechanical deflection of the duodenal section, which recently has been emphasised by Kundrat,[27] Schnitzler,[28] Albrecht,[29] Müller.[30]

From the recorded material, we may ætiologically differentiate seven kinds of acute gastric paresis:—

1. Gastric paresis after errors in diet (paresis ab ingestis, acute over-exertion of the stomach). To this group belong the observations of Hilton Fagge,[31] A. Fraenkel,[32] Kirch,[33] Kuttner,[34] A. Heine,[35] and the author.[36] The cases of Fagge and Kirch terminated fatally; but it must not be excluded that in either case the acute gastric dilatation was caused by a mechanical deflection in the previously mentioned sense. The grave clinical picture described by Kirch points at least with great probability to such a cause.

2. Gastric paresis after severe infectious diseases, such as the cases

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[27] Kundrat, reviewed in the Wiener medicinische Wochenschrift, 1891, No. 8.

[28] Schnitzler, Wiener klinische Rundschau, 1895, No. 37-38.

[29] Albrecht, Inaug.-Dissert., 1899; Virchow's Archiv, 1899, Bd. 156, S. 285.

[30] P. Müller, Deutsche Zeitschrift für Chirurgie, 1900, Bd. 56, S. 486.

[31] Hilton Fagge, Guy's Hospital Reports, 1873, Bd. 18; reviewed in Virchow-Hirsch's Jahresbericht, 1873, Bd. 2, S. 83.

[32] A. Fraenkel, Deutsche medicinische Wochenschrift, 1894, No. 7.

[33] Kirch, Deutsche medicinische Wochenschrift, 1899, No. 33.

[34] Kuttner, bei Lindner-Kuttner, *l.c.*, S. 79.

[35] A. Heine, Hospitalstidende, 1899. Quoted from Archiv für Verdauungskrankheiten, Bd. 6, Heft 1, S. 86.

[36] Boas, Deutsche medicinische Wochenschrift, 1894, No. 8.

reported by Albu[37] (development after scarlatina) and by Hood[38] (after pneumonia complicated with suppurative pericarditis).

3. Gastric paresis after exhausting chronic diseases. Cases reported by Schultz,[39] A. Fraenkel,[40] Brown,[41] Kelynack,[42] Riedel.[43] A case recently reported by Brown,[44] developed in a patient with chronic nephritis.

4. Gastric paresis after traumatism. Observations by Erdmann,[45] Appel,[46] Box and Wallace,[47] and Rosenheim;[48] the latter's case is very characteristic.

5. Gastric paresis after laparotomies (Köberle,[49] M. Gross,[50] Hunter,[51] Grundzach,[52] Robson,[53] Fenger,[54] Kehr,[55] Stieda.[56] These cases show a partially analogous similarity to the numerous observations of grave, post-operative intestinal paralysis, the ætiology of which is still entirely unexplained, the same as the gastric paresis in question.

6. Chloroform narcosis alone, as observations of Morris, Riedel, and others indicate, may lead to development of an acute gastroparesis, probably by way of central paralysis.

7. Far more characteristic are the cases of acute gastroparesis which are caused by mechanical kinking of the pylorus or the jejunum or through acute obstruction of the pylorus (gall-stones perforated into the pylorus). Particularly the recent observations of Kundrat, Schnitzler, Albrecht, P. Müller have exhibited partly torsions of the mesenterium, partly compressions of the duodeno-jejunal section by the arteria mesaraica superior as causes of such kinking.

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[37] Albu, Deutsche medicinische Wochenschrift, 1896, No. 7.

[38] Hood, The Lancet, December 19, 1891, P. 1389.

[39] Schultz, Jahrbuch der Hamburger Krankenanstalten, 1890, Leipzig, 1893, S. 145.

[40] A. Fraenkel, *l.c.*

[41] Brown, reference in Deutsche medicinische Wochenschrift, 1893, S. 1344.

[42] Kelynack, Medical Chronicle, May, 1892.

[43] Riedel, Penzoldt-Stintzing's Handbuch der speziellen Therapie, 1896, Bd. 4; Abtheilung 6 B., S. 133.

[44] Brown, The Lancet, 14 October, 1899.

[45] Erdmann, Virchow's Archiv, 1868 Bd. 43.

[46] Appel, Philadelphia Medical Journal, August 12, 1899. (Refer also to Stieda 20).

[47] Box and Wallace, The Lancet, June 4, 1898, P. 1538.

[48] Rosenheim, Berliner klinische Wochenschrift, 1897.

[49] Köberle, Société médicale de Strassbourg, 1873. Quoted from Bouveret.

[50] M. Gross, Leçons de clinique chirurgic., Nancy, 1873. Quoted from Bouveret.

[51] Hunter, New York Medical Record, 1889.

[52] Grundzach, Wiener medicinische Presse, 1879, No. 43-44.

[53] Robson, The Lancet, 1900, Vol. 1, page 832.

[54] Fenger, Clinical Revue, 1899. Quoted from Robson.

[55] Kehr, Langenbeck's Archiv, 1899, Bd. 58.

[56] Stieda, Zeitschrift für Chirurgie, 1900, Bd. 56, S. 201.

As the acute gastroparesis deviates in many parts of the symptomatology from the chronic form, we consider it expedient to give a brief collation of the diagnostically important points, according to the observations reported.

The *clinical picture* varies according to the cause and extent of the process: milder where acute paresis develops in connection with a previously existing atony through overloading of the stomach; graver, even life-threatening, where real and complete mechanic deflections are present, as is the case in at least a number of so-called post-operative gastric dilatations. In milder cases, manifestations of acute dyspepsia are associated by a single or repeated vomiting, after which either at once recovery ensues, as in the case of A. Fraenkel, or the symptoms of a subacute stasis insufficiency may last for a few weeks, as I observed. In grave cases *the prevailing symptom is the exceedingly violent and partly bilious, partly brownish or blackish (blood) vomiting. The vomit is by no means fæculent.* The abdomen is, in the majority of cases, not very much distended; usually a high degree of tympany is not present. Pain was observed in several cases, but is not a constant symptom, or is at least less conspicuous than the other symptoms.

The patient alway suffers from thirst. Temperature, as a rule, is not increased, occasionally even subnormal; pulse, rapid and small, respiration frequent (agitated) and superficial. As a rule, flatus and evacuation of fæces are entirely suspended until a turn for the better takes place.

Diagnosis.

The *diagnosis* of the acute gastric insufficiency is easy in the milder cases, especially if an ætiologic factor is evident, but more difficult in the peracute cases, post-operative cases, especially after laparotomies. In such cases, the diagnosis is not often recognised. The differentiation from acute peritonitis may be very difficult. It is true, the vomiting in the latter is never very extensive, blood vomiting does not occur, symptoms of real obstruction are not present, while, on the other hand, fever, though moderate, points to an inflammatory process. These, however, are only comparatively decisive diagnostic signs. Only the direct proof of an acutely developed dilatation of the stomach by physical examination will be the deciding factor. If *bilious* vomiting continues, an occlusion beyond the pars descendens duodeni may be assumed with a certain probability, and, based upon the recent pathologic-anatomical findings, an arterio-mesenteric obstruction of the jejunum will be probable. The diagnosis is much more simple after operations which did not touch the peritoneum. In such cases, in consideration of the fact that post-operative dilatations are not a rare occurrence, the diagnosis cannot be easily mistaken.

Treatment.

As to prophylaxis, first of all the principle must be established that in all laparotomies as well as in other operations more attention must be given to the condition of the stomach than had been done to the present time. If an atony is existing, the administration of food after an operation must be very cautious. Riedel[57] applies even lavage and hypodermic injection of morphine in all cases of post-operative vomiting which continues for more than twenty-four hours, and claims to have saved at least six patients who were threatened with gastric dilatation.

In cases of imminent or existing gastric paresis, all feeding by the mouth must be stopped and food may be given only by the rectum. Subcutaneous salt infusion may be indicated. Lavage of the stomach is urgently to be recommmended for the relief of the vomiting as well as for the loosening of any mechanical obstacles. Simultaneously treatment with opium and, on account of the latest experiences in cases of ileus, with atropine (doses of 0.001 to 0.005) should be instituted (Batsch[58]).

If mesenteric occlusion of the intestines is suspected, systematic abdominal position should be taken, according to Schnitzler's and Müller's favourable experiences.

In extreme cases, gastro-enterostomy (a case cured by Kehr) should be tried, though the prospects for a cure are exceedingly doubtful, the condition of the patient being usually hopeless.

(B) Chronic Gastric Insufficiency.

Most frequently we have to deal with the second chronic form of gastric insufficiency, to which we now turn in the following:—

Diagnosis.

The diagnosis may be divided into two parts: first, the characteristics of the mechanic obstacle must be stated; second, the causes must be elucidated.

For the ascertainment of the mechanic obstacle there may serve (*a*) the peculiar subjective symptom-complex, (*b*) the demonstration of the kind and degree of the functional diminution, whereby the establishment of the gastric borders and capacity is of great importance.

The following symptoms have to be considered for the diagnosis of a motor insufficiency of the second degree:—

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[57] Riedel, quoted by P. Müller, Deutsche Zeitschrift für Chirurgie, 1900, Bd. 56, P. 209.

[58] Batsch, Münchener medicinische Wochenschrift, 1900, No. 15 and 27.

(*a*) *Subjective Signs.*

They include, first, all manifestations on the side of the afflicted organ itself; second, the general effects of the affection. The local complaints are connected with the act of digestion, and consist of pressure and fulness after meals, distension of the stomach and intestine, pyrosis or cramp-like pains, radiating towards the chest or the back. These symptoms are the expression of an attempt to propel the contents, which are too large in comparison to the existing strength, through the pylorus. If this succeeds to a certain degree, rest takes place after the passage; but gradually it succeeds less frequently and less completely, the contents stagnate, the power of absorption sinks, the stomach works more spasmodically and evacuates the stagnant masses towards the locus minoris resistentiæ, *i.e.,* upward—in short, vomiting takes place. If the patients have recognised the beneficial influence of vomiting, they try to bring it about by manipulations of every kind. As the vomiting represents, so to say, the last desperate attempt of the overexerted organ, it occurs only some hours after the main meal, either during the night or early in the morning. The vomit consists of large masses of very sour or bitter-tasting fluids, mixed with comparatively small quantities of solid food-substances. It contains not only remnants of the last meals, but, according to the degree of motor insufficiency, remainder of food which had been ingested days, and in some cases even weeks, previously. *If the patients can state with certainty that after regular ingestion of food they repeatedly vomited food in the morning before breakfast, this symptom alone is already sufficient to diagnose a mechanical insufficiency of the second degree.*

The condition of *appetite* and *thirst* is also important. The former is good as long as the peristaltic function is tolerably good; along with the moment of establishment of stagnant masses, the appetite becomes gradually weaker, to drop to nil. In cases of beginning stasis insufficiency, I often observed intercurrent bulimia, which occasionally occurs also later, though much less intense. The appetite is in a reversed relation to thirst. The greater the latter, the less the former. The degree of thirst, measured by the dryness of the tongue, is a fair measure for the degree of the insufficiency. Apparently paradoxical is the experience that in spite of a stomach filled to the cardia, thirst is the most violent; it may be explained that the greater the pressure of the fluid column, the slower and the more incompletely the ingesta are propelled into the duodenum.

Through the existing retention of fluids in the stomach, the intestinal function is influenced unfavourably; only a few hard and dry scybala are evacuated. The abnormal fermentation, present already in the stomach, finds still more favourable conditions for formation in the intestine.

The final products of fermentation, the gases, combine with those formed in the stomach, and, on account of the resulting enormous distension of the gastro-intestinal walls and of the specific influence upon the intestinal nerves, cause a large number of complaints, such as violent colics, tenesmus, headache, sensation of pressure in the abdomen, hæmorrhoids, etc.

The peristaltic function of the intestine is likewise a good criterion for the type and degree of the process. The weaker it is and the oftener and the stronger drastica are necessary, the more advanced the affection. Temporary diarrhœa is rare, and it is not certain whether it is simple stercoral diarrhœa, or an irritative effect of the decomposed masses which enter the intestine. The appearance of spontaneous copious stools is a favourable sign for the efficiency of the treatment.

Among the general manifestations, the *nutritive disturbances* are of greatest importance. The same are dependent on the one side upon the loss of appetite, on the other side upon loss of water through vomiting, and finally upon the diminished absorption. The loss of weight is considerable, amounting in severe cases to ten or even twenty kilos and more in the course of a few weeks, whereby first the subcutaneous fatty tissue, and later the muscle albumen is affected. Simultaneously—effect of deficient absorption of fluid—the skin becomes dry, scaly, cool, wanting the normal turgescence. The patients complain of rapidly tiring, dyspnœa, inclination to sleep, numbness in their finger-tips and toes, frequently in their calves, occasionally in the upper limbs, rarely in the facial muscles, and finally tetany (see below).

(*b*) *Objective Symptoms.*

We have to turn our attention to inspection, palpation, percussion, auscultation, transillumination, tube-palpation, and finally, examination of the gastric contents in regards to the chemical, motor, and absorbent function.

Inspection, especially as emaciation is almost regularly pronounced, yields important hints as to position and size of the organ. If the patient is in a recumbent position, and you are standing at his head, you may see the greater curvature in its entire extension, more or less below the umbilicus. Occasionally peristaltic[59] or, rarer, anti-peristaltic[60] waves may be observed in this region.

Somewhat more exact results are given by *percussion*, or percussion combined with palpation (palpato-percussion). Referring as to its technique to the General Part (pp. 88, 92), and to the statements in the section on Insufficiency of the First Degree, we limit ourselves to the fact that we may determine fairly well the greater curvature through percussion, particularly through the splash-sound (p. 86), produced by percussion, either when the stomach is full, or artificially filled with matter for this purpose, and the intestine is empty. If the stomach is empty and the intestines full, the determination of the borders is more uncertain. The determination of the largest horizontal and vertical diameters (p. 93) by percussion may succeed under favourable circumstances, and yields a useful picture of the size of the stomach. By inflation or transillumination of the stomach we get better results. [Röntgen rays.] Walter Michaelis,[61] on careful percussion of the stomach, found in many cases of motor insufficiency an enlargement of the stomach toward the right side, and attributes to it a certain importance for the diagnosis of the motor gastric insufficiency.

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[59] Kussmaul, Volkmann's Sammlung klin. Vorträge, 1880, No. 181.

[60] Vgl. Schütz, Prager med. Wochenschrift, 1882, No. 11; A. Cahn, Deutsch. Archiv für klinische Medicin, 1884, Bd. 35, S. 402; J. Glax, Pester medic. chirurg. Presse, 1884.

The best method, and in my opinion sometimes the only decisive one, is the *palpation of the stomach*, but to be sure only—and this may be emphatically stated—in cases of pyloric stenosis which, as already mentioned, are much more prevailing in number than the atonic dilatations. In such cases two kinds of motor phenomena may be observed: first, and that only rarely, a tonic spasm (pylorism), extending only over the pylorus, which can be distinctly felt for a few moments by the hand put thereon, in order to disappear and appear anew, then a tonic contraction of the fundus, which occurs in various degrees of intensity, sometimes a slight hardening of the stomach, hardly felt by the patient, sometimes as a rigid and stiff contraction, which I designate according to Nothnagel's apt nomenclature, as "*stiffening of the stomach*," and finally as the most pronounced form of the spasmodic gastric contractions, the visible and palpable *spasmodic* peristalsis of the stomach. There is no doubt about the significance of the latter—it is an unmistakable sign of an obstacle situated on the pylorus or beyond the same. But the visible gastric tetanus is a comparatively rare phenomenon and symptom of the most pronounced pyloric stenosis.

[A most excellent and very pronounced picture of this gastric tetanus came under my observation as late as March, 1906. The patient was a woman of twenty-five years of age. The initial cause of the pyloric stenosis was continuous vomiting during the whole period of vomiting, which resulted undoubtedly in the formation of erosions. Patient was to be operated on for "floating kidney" in May, 1905, and on opening the abdomen a tumour in the pyloric region was found; the operation was not continued, as the tumour was supposed to be an inoperable cancer of the lesser curvature. All the symptoms, when I saw the patient, indicated a benign obstacle, and not a malignant tumour. There existed hyperchlorhydria of over 80; yeast and sarcinæ very abundant. The relief by lavage, followed by introduction of olive oil, was most pronounced. Patient was exceedingly emaciated, had lost about

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[61] Walter Michaelis, Zeitschrift für klin. Medicin, 1898, Bd. 34, P. 241.

60 pounds (from 150 to 90); when the stomach was empty, no pain existed. The simulated tumours disappeared under the palpating hand while lavage was performed. Patient died of inanition, which first showed itself in decrease of urine, delirium, and later in coma. Rectal feeding and introduction of salt water were unavailing. This patient, if operated on in time, would have had an excellent chance of recovery.]

On the other side the beginning, just noticeably stiffening of the stomach is a symptom which elucidates in many cases, even in a very early stage, the diagnosis of pyloric stenosis.

I will, therefore, not fail to give a few practical hints for elucidating the stiffening of the stomach. Above all, the examination should be made on a full stomach, or rather over-filled stomach, expediently about three to four hours after an abundant dinner. Seize the fundus of the stomach with the whole hand and rub it with the hand, kept cold by dipping in cold water. You will soon notice a hardening of the fundus section, lasting only a short time, but gradually increasing, vanishing very soon, then reappearing anew after a few seconds. The practitioner will find it expedient to practise this phenomenon, first, in cases of pronounced pyloric stenosis, and he will soon be able to ascertain it in less pronounced cases. It may be well known that by irrigation with ether or by faradisation, the phenomenon may be likewise elucidated or fortified.

Furthermore, we succeed by means of transillumination, inflation with air or CO_2, or tube-palpation, in establishing the position of the greater curvature, and in many cases also the size and position of the stomach.

The *auscultation* has a diagnostically inferior importance; you hear, on shaking the abdomen of the patient, even if the patient moves but slightly sideways or forward, a splashing noise, "as if fluid in a half-full cask is moved to and fro" (succussion sound).

Moreover, you hear, putting your ear to the stomach region, various bubbling, hissing, and squirting noises, which are observed to a greater extent than normally and differentially from the healthy stomach, also in the empty stomach; but they have nevertheless a diagnostically inferior significance.

The *examination of the stomach-contents* yields, even looked at quite cursorily, exceedingly important hints for the diagnosis of stasis insufficiency. By this procedure alone we learn whether the stomach empties itself punctually, tardily, or not at all. In order to elucidate this fact, various ways may be taken. I recommend the following two as the most expedient, as they will surely establish the condition. The first one is the administration of Leube-Riegel's test-meal (a plate of soup, a roll, a beef-steak); after seven hours, removal of the contents. The normal stomach will be empty; the slightly insufficient stomach will contain only remnants of the last meal; the highly insufficient stomach will contain copious remnants, partly decomposed, and also remnants from previous meals. If you will decide whether stagnant contents are present in the stomach in the morning before partaking of any food whatever, the patient eats the above-mentioned "test-supper" at 8 P. M. If the stomach is normal or atonic, no remnants are left in the morning; in cases of insufficiency of the second degree, however, more or less copious remnants, characterised by a state of decomposition, are found. For purposes of general practice, it may suffice to examine the stomach without this particular preparation, and you will rarely miss, in cases of stasis insufficiency, remnants of the meals taken during the previous day or evening. Nevertheless, the test-supper is a considerably surer method.

In examining the stomach-contents, we have first of all to consider the quantity, macroscopical appearance, the smell, abnormal additions. A more exact examination has to deal with the chemical and microscopical condition of the contents.

The *quantity* considerably surpasses the masses found in other gastric affections, though as a matter of course, it varies greatly according to the type and degree of the affection. The appearance is especially remarkable in the fact that it shows the well-known three layers, caused by marked gas fermentation, whereby the specifically lighter ingredients of the contents are forced to the top. In pronounced cases "gas bubbles similar to the pearls in a glass of champagne into which bread globules were dropped" (Ewald[62]) are observed.

In the stomach-contents remnants of previous meals, even remnants of meals ingested days, weeks, or months previously, are found. Particularly the seeds and peels of fruit remain easily and for an exceedingly long time in the relaxed gastric folds.

The *smell* of the stagnant gastric contents is likewise very characteristic; it smells, especially if hydrochloric acid is present, pungent sour, like must or badly fermented beer, and exhibits, especially if fresh, the odour of H_2S.

To demonstrate the latter, it is advisable to express a part of the stomach-contents in a large medicine-bottle which can be well corked. After letting it stand for some time, H_2S may be demonstrated either by the smell; or, still more certainly, by a strip of filter-paper, moistened by dipping it into a solution of acetate of lead and caustic potash, and suspended from the cork over the fluid. If HCl is absent, the stagnant contents has a more fatty, sour, sometimes nauseous smell [vinegar smell]. Addition of pus (see Chapter on Carcinoma) gives the stomach-contents a highly fœtid smell.

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[62] Ewald, Klinik der Verdauungskrankheiten II, 3 Aufl., P. 274.

Occasional admixtures are those of bile and pancreatic juice. *Constant* presence of bile, fresh or decomposed, speaks for stenosis in the pars descendens duodeni.

The *chemical* and *microscopical* examination of the gastric contents, whatever the ætiology of the mechanical insufficiency may be, shows usually equal results: the accumulation of intensely sour masses in the stomach, whether conditioned by HCl, or combined, or finally fermentative acids. *The total acidity is accordingly usually increased considerably.* The amount of organic acids depends upon various factors, especially on the kind of the ingesta, the degree of stagnation and the condition of the gastric glands. It has been claimed that in some cases abnormal decompositions, particularly production of acetone and of related substances, have been observed (v. Jaksch,[63] Lorenz,[64] v. Noorden[65]). The presence of acetone in the gastric contents has been disputed by Savelieff,[66] relying upon recent investigations. The carbohydrates, too, if retained in the stomach for a long time, may experience decomposition, producing carbonic acid, hydrogen, marsh gas, oil-forming gas, etc.

On microscopic examination among numerous bacteria, particularly two kinds of fungi are observed—*yeast in sprouting,* and *sarcina;* they accompany one another frequently, but not regularly. The sarcinæ, the biology of which has been exhaustively investigated by Falkenheim,[67] Oppler,[68] Stubenrath,[69] and Ehret,[70] are generally observed in the well-known bale-form, and also in form of separate bunches, clouds (Ehret), or package-like groups. The sarcinæ, like the yeast fungi, find the most favourable conditions for propagation in a medium containing HCl. According to Ehret's and my observations, sarcinæ are found in cases of stasis insufficiency, not only in the stomach-contents, but also in the fæces; in such cases, they are occasionally—but not always, as Ehret claims—imbibed with bile.

Are the sarcinæ of diagnostic importance? This question must be decidedly affirmed, as far as you do not pay particular attention to a few bales of sarcinæ which occasionally may be swallowed with the food and may also be mixed with the gastric contents of healthy persons, but as far as you observe the presence of numberless packets in every field of vision.

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[63] v. Jaksch, Zeitschrift für klinische Medicin, 1884, Bd. 8, Heft 1 u. 2.

[64] Lorenz, Zeitschrift für klinische Medicin, 1891, Bd. 19, S. 19.

[65] v. Noorden, Lehrbuch der Pathologie des Stoffwechsels, Berlin, 1893, S. 249.

[66] Savelieff, Berliner klinische Wochenschrift, 1894, No. 33.

[67] Falkenheim, Archiv für experimentelle Pathologie und Pharmakologie, Bd. 19, S. 339.

[68] Oppler, Münchener medicinische Wochenschrift, 1894, No. 24.

[69] Stubenrath, Das Genus Sarcina, mit besonderer Berücksichtigung der Magensarcine, München, 1897.

[70] Ehret, Mittheilungen aus den Grenzgebieten, 1897, Bd. 2, S. 744.

The latter is the case, according to my extended experience, exclusively in stasis insufficiency and most frequently when HCl is present. If large quantities of lactic acid are present, the finding of sarcinæ—and I emphasise this, in spite of Ehret's statement to the contrary, is exceedingly rare. Sarcinæ, however, are found in cases in which there is no free hydrochloric acid, but abundant combined hydrochloric acid, and lactic acid is absent. If after total disappearance of HCl, lactic acid formation gradually takes place, sarcinæ, present hitherto, usually begin to disappear.

On the other side, in cases of abnormal lactic acid formation, *lactic acid bacilli,* described first by Oppler and myself, are found. We shall revert to them in the Chapter on Carcinoma of the Stomach. Frequently with either form of fermentation numerous fatty acid crystals, bunched together in the shape of rosettes or stars, are found.

Moreover, the microscopic finding varies and is characteristic only in so far that in cases of lactic acid fermentation, in which the digestion of albumen is lessened, more muscle-fibres and less unchanged amylum grains are found; whereas in the presence of hydrochloric acid muscle-fibres are shown in but small quantities, and very great masses of well-preserved starch remnants may be demonstrated by the iodine test.

A frequent symptom of the gastric contents in stasis insufficiency is the presence of combustible gases in the stomach, among which particularly hydrogen in large quantities was demonstrated (Ewald,[71] McNaught,[72] G. Hoppe-Seyler,[73] F. Kuhn[74]). As was to be supposed, abundant carbonic acid was found also. To Hoppe-Seyler and also Kuhn belongs the merit of having pointed out the importance of the differentiation of the presence of plain air from that of pathologic fermentation for the diagnosis and therapeutics of the gastric diseases. In accordance with each other, these authors demonstrated that the production of said gases is not inhibited by the presence of large amounts of hydrochloric acid. But Kuhn, after extended experiments, demonstrated the inhibiting influence of other antiseptic substances, and that not only in the test-tube, but also in persons suffering from ectasia. We shall revert to this subject in the section on treatment. I myself,[75] and later Zawadsky,[76] could demonstrate that decomposition of albumen takes place in the ectatic stomach, in so far that in a large number of cases of grave motor insufficiency, particularly fresh cases which were treated insufficiently or incorrectly, one of its final products, H_2S, could be repeatedly detected. In these cases, too, free HCl was present, partly in normal or subnormal quantities, partly in supernormal quantities. We may conclude from the mentioned investigations that the *mechanical obstacle of the gastric digestion represents the chief cause of the fermentation,* and that the presence or absence of hydrochloric acid is but of inferior importance.

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[71] Ewald, Archiv für Anatomie und Physiologie, 1874, S. 217.

[72] McNaught, British Medical Journal, 1890, No. 1522.

[73] G. Hoppe-Seyler, Verhandlungen des Kongresses für innere Medicin, 1892, S. 392; Deutsches Archiv für klinische Medicin, 1892, Bd. 50, S. 82.

[74] F. Kuhn, Zeitschrift für klinische Medicin, 1892, Bd. 21, S. 572; Deutsche medicinische Wochenschrift, 1892, No. 49 and 50.

[75] Boas, Deutsche medicinische Wochenschrift, 1892, No. 49; Centralblatt für innere Medicin, 1895, No. 3.

[76] Zawadsky, Centralblatt für innere Medicin, 1894, No. 50.

Such is the condition of the *stagnant* stomach-contents in the grave form of insufficiency. There occurs in such cases still another anomaly of the gastric function, the so-called excessive secretion of the gastric juice (supersecretio acida, gastrosuccorrhœa, designated by the French as *Reichmann's disease*) (see Appendix, p. 532).

The quantity of the contents in the stomach before partaking of food, is simultaneously a good measure for the extent of the motor insufficiency; the more copious the retained matter, the greater the *insufficiency* (see methods of Mathieu and Rémond, page 154.

We possess also a comparatively useful measure for the degree of the insufficiency of the secretion of urine; we may distinguish three stages:

First stage: amount of urine for twenty-four hours is 1500 to 1000 cubic centimetres.

Second stage: amount of urine for twenty-four hours is 1000 to 500 cubic centimetres.

Third stage: amount of urine for twenty-four hours is below 500 cubic centimetres.

The urine is often alkaline, especially in those cases of dilatation in which, on account of permanent mechanic irritation, a continuous secretion of gastric juice takes place. The urine often shows milk-white precipitates of phosphates, which on microscopic examination usually manifest themselves as triple-phosphates—the well-known coffin-lid form—rarely as *magnesium phosphate.*[77] The specific gravity of the urine is usually considerably increased. The chlorides are decreased in grave cases of insufficiency of both benign and malignant origin.

Other anomalies in the urine are *acetone and diacetic acid;* it is claimed that albumosuria may occasionally occur in cases of ulcerative processes in the gastro-intestinal tract. These deviations have not been investigated enough to be useful for clinical purposes.

It is also undoubted that absorptive disturbances are present in cases of stasis insufficiency, but as to their extent we are directed more to indirect evidence.

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[77] Ebstein, Deutsches Archiv für klinische Medicin, Bd. 31, P. 203.

Diagnosis of Some Important Types of Gastric Insufficiency.

The above-discussed symptoms serve to ascertain the diagnosis of stasis insufficiency, and the special diagnosis of the type of the insufficiency was partly discussed. In some cases the general diagnosis of a grave motor disturbance will have to satisfy; in other cases the diagnosis may be advanced to the ascertainment of a mechanic obstacle, and under the most favourable circumstances it may be possible to establish even the nature of the obstacle. The causes being very much varied, it is excluded to exhaust all possibilities. It may suffice to keep in mind the clinical picture of the most frequent and practically most important types of stasis insufficiency. Among them are: (1) muscular stasis insufficiency; (2) benign cicatrised pyloric stenosis; (3) pylorospasm; (4) hypertrophic pyloric stenosis; (5) carcinomatous pyloric stenosis. The latter will be discussed in the Chapter on Cancer of the Stomach.

(1) *Muscular Stasis Insufficiency.*

This may be caused by paralysis of the neuro-muscular apparatus, or by a specific affection of the muscularis (fatty and colloid degeneration), or by hyperplastic processes in the muscularis (Linitis plastica, Brinton), or by destruction of the muscularis through toxic processes (cauterisation, corrosion, etc.), or, finally, by carcinomatous or sarcomatous degeneration migrating into the muscularis.

Among all these types the paresis of the smooth musculature dependent on the primary atony possesses the clinically greatest interest. The same are, however, as mentioned above (p. 489), by no means so frequent as a chronic condition, as formerly was supposed.

The diagnosis of a chronic stasis insufficiency dependent upon a myasthenic condition will probably never be made with any degree of certainty. How could kinks or adhesions, stenoses or compressions of the pylorus or duodenum be excluded with certainty or even probability? Perhaps a continuous absence of gastric stiffening or of visible or palpable peristalsis might speak more for muscular insufficiency than for a mechanical obstacle on or in the neighbourhood of the pylorus. But as we have to deal, at least in cases not much advanced, with but temporary motor phenomena, their absence is not an absolute evidence against the possibility of stenosis. Evidence of the absence of an organic obstacle could be produced only by introduction of the tube into the duodenum, although even this measure would not entirely exclude the existence of an impediment in the sphere of the duodenum. Moreover, the methods serving for this purpose (Kuhn, John C. Hemmeter[78]) are only in the beginning.

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[78] Hemmeter, Diseases of the Stomach, Philadelphia, 1902, P. 56 to 59.

The diagnosis of muscular paralysis through benign organic processes (hypertrophic muscularis, fatty colloid, degeneration, Linitis plastica) is still awaiting an exact scientific base. The diagnosis of the carcinomatous and sarcomatous degeneration coincides with the diagnosis of the original diseases.

(2) *Cicatrised Pyloric Stenosis.*

This type is formed by the cicatrised stenosis which develops in connection with an ulcer situated at, or near to, or below the pylorus. A quite similar picture may be produced also by corrosion of the pylorus through caustic poisons.

The diagnosis of the pyloric stenosis developing after an ulcer is easy, as soon as the history points with certainty or great probability to this ætiology, *i.e.*, when hæmatemesis or malæna, in conjunction with preceding evident ulcer symptoms, is followed a shorter or longer time afterward by the symptoms of a gradually increasing motor insufficiency. Such cases form the type of the ectasia, formerly so-called, to the clinical picture of which we will not revert again. The previously described contraction phenomena are most pronounced in these cases. If certain ulcer symptoms cannot be found in the history, we may perhaps succeed in supposing an organic pyloric stenosis; its causes (carcinoma, ulcus carcinomatosum, benign hypertrophy, adhesion, etc.) may be elicited under favourable circumstances, otherwise they must be left undecided.

The differentiation of a cicatrised pyloric stenosis from that caused by perforation of a gall-stone (calculus stenosis) may become very difficult. It is true, in a few cases the diagnosis was made by others and myself, but frequently it will be missed. We may suggest it under the following conditions: when cholelithiasis was positively observed, if possibly stones were found, when the process manifestly followed subsequently and lasted for several years, when absolutely no symptoms of gastric ulcer had existed or still exist, and when the patient is not at all in the age of development of ulcers.

The differential diagnosis between cicatrised stenosis of the pylorus and that of the duodenum is perhaps possible as regards the pars descendens of the latter. In such cases, the constant presence of bile, and perhaps also of pancreatic juice, would speak in favour of the situation of the obstacle below or near the papilla Vateri. If, furthermore, an ulcer hæmorrhage can be positively ascertained from the history, the ætiologic connection with the same gains in probability. Under similar certainly rare circumstances, I succeeded once in making the diagnosis. The other possibilities which may come under differential diagnostic consideration cannot be discussed here. A differentiation of a *suprapapillary* cicatrised stenosis—an exceedingly rare occurrence—from that of the pyloric section has hitherto not been positively made. But Czygan[79] succeeded in diagnosing a carcinoma situated in the upper portion of the duodenum on account of the splash sounds which were demonstrable particularly between the tumour and the costal arch. The same physical conditions may, as a matter of course, also be of importance as to benign stenosis situated similarly in the duodenum.

As to differentiation of the cicatrised pyloric stenosis from the hypertrophic one, compare farther below.

(3) *Spastic Pyloric Stenosis.*

If erosions or fissures are situated at or near the pylorus, the pylorus, particularly under the influence of unsuitable food, falls into a spasmodic condition which manifests itself especially at the acme of digestion, by violent gastralgia, sometimes ending with vomiting. These spasms may be continuous or may occur periodically, and may lead finally to a spastic occlusion of the pylorus and retention of food. We already know the picture of spastic pyloric stenosis in its chief points from Kussmaul's celebrated description. But the importance of the spastic pyloric stenosis has only lately been brought to the light through the investigations of Doyen, Carle and Fantino, P. Cohnheim, [D. D. Stewart], and others.

The clinical picture of spastic pyloric stenosis shows the following conditions: the stenotic picture proper is preceded, as the history manifests, by the above-mentioned symptoms of pylorospasms, which, of course, cannot always be differentiated from those of gastric ulcer. Subsequently there follows retention of gastric contents, which manifests itself through violent and spasmodic pains, most pronounced in the pyloric region, and followed by vomiting of intensely sour *decomposed* masses. Afterward, relief until the following day or the day after, when the same cycle of disturbances begins anew. All other previously mentioned typical symptoms associated with stasis insufficiency follow, namely, rapid emaciation, oliguria, severe constipation, increased thirst, and usually well-preserved appetite, even bulimia.

The diagnosis is made from the following symptoms. The pyloric region is frequently tender on pressure; the stomach may be dilated, but such is not always the rule. In the pyloric region tonic contractions may be sometimes observed, and more or less pronounced stiffening of the stomach may be palpated on the fundus on repeated and careful examination. The examination of the gastric contents after the test-supper shows either stagnation (in such cases increased secretion of HCl, sarcinæ, yeast, fatty acid crystals, considerable fermentation), or instead of stagnant gastric contents, the presence of gastric juice in pathologic quantities (more than 100 cubic centimetres). Occasionally these two conditions happen to alternate; as a rule, the cure of the process passes through the intermediate stage of supersecretion, which under favourable conditions gradually disappears. But even then a motor disturbance can be demonstrated by means of the methods of v. Leube and Mathieu-Rémond.

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[79] Czygan, Archiv für Verdauungskrankheiten, 1897, Bd. 3, P. 82.

The prognosis of the spastic pyloric stenosis is, in contrast to the organic stenosis, according to my experience, often favourable. To be sure, there are occasional relapses.

(4) *Hypertrophic Pyloric Stenosis.*

The pylorus may become hypertrophic either alone or in conjunction with the fundus region under very varied conditions, which ætiologically can hardly be compared to one another. The best-known form is the gastric cirrhosis described by Brinton (Linitis plastica); Nothnagel[80] gives the report of one case, interesting on account of its complication with pernicious anæmia. Furthermore, to this form belongs the "gastrique chronique avec sclérose sousmuqueuse" described by French writers. Very important is the third form, which may be congenital, but may, according to recent literature, be frequently observed in the adult also, namely, hypertrophic pyloric stenosis in its stricter meaning. After this had already been closely studied by Lebert,[81] it was later observed and described by Nauwerck,[82] Tilger,[83] Rosenheim,[84] Einhorn,[85] Hemmeter,[86] and myself.[87]

Hypertrophic pyloric stenosis of the adult (which alone will be described here) may likewise develop under a great variety of conditions, for instance, as a residue of an ulcer of the pylorus, after cauterisation and corrosions, etc.; but as Lebert, Hemmeter, and myself have shown, it may be caused by a chronic gastritis, and it seems that this represents the most frequent cause for development of a hypertrophic pyloric stenosis. I have therefore suggested designating this type of stenosis as a "stenosing gastritis," a name adopted also by Hemmeter.

Stenosing gastritis is generally a disease occurring at a comparatively young age. Tilger, however, reported also cases in persons at the age of over fifty and sixty years. It must not be forgotten that patients sometimes come under observation only many years after beginning of the affection or that diagnosis is made only then.

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[80] Nothnagel, Deutsches Archiv für klinische Medicin, 1879, Bd. 24, S. 353.
[81] Lebert, Die Krankheiten des Magens, Tubingen, 1878, S. 525.
[82] Nauwerck, Deutsches Archiv für klinische Medicin, 1878, Bd. 21, S. 574.
[83] Tilger, Virchow's Archiv, 1893, Bd. 132, S. 290.
[84] Rosenheim, Berliner klinische Wochenschrift, 1894, No. 39.
[85] Einhorn, New York Medical Record, January, 1895.
[86] Hemmeter, Diseases of the Stomach, Second Edition, P. 613.
[87] Boas, Archiv für Verdauungskrankheiten, 1898, Bd. 4, P. 47.

The clinical picture of stenosing gastritis is coincident with that of chronic gastritis, and obtains its peculiarity only when the motor disturbances commence.

The same may in the beginning manifest themselves only by a feeling of pressure and fulness, by eructation and pyrosis; the symptoms gradually increase and become more pronounced; there occurs stagnation and along with it vomiting, sometimes of small quantities, sometimes of copious masses, sometimes daily, at other times at certain shorter or longer intervals.

The general health begins then to suffer, weight decreases, diuresis is checked, more or less obstinate constipation shows itself; in brief, there develops the picture of chronic stasis insufficiency. The pyloric section may be only thickened, or may turn into a smooth, regular tumour. The fundus of the stomach may be in normal position or may be considerably displaced (as in one of my observations), whereby also the pylorus drops unless fixed by adhesion. The gastric capacity may be increased, normal or even subnormal.

The *diagnosis* of the stenosing gastritis is made from the long duration, the numerous remissions and exacerbations; furthermore, the chronic gastritis on the one side and the stenotic symptoms on the other side. The former is based upon the fact of the decreased secretion or (as in my observation) of the absolute absence of hydrochloric acid and ferments. The stenotic symptoms exhibit the characteristics of stasis insufficiency as described in the general part. At the stage of HCl secretion lactic acid may be absent, but may be present in the further course, as in cases of carcinoma. The lactic acid bacilli, described by Oppler and myself, are also found in large numbers in the stomach contents. If the pylorus can be palpated as a purely thickened and symmetric cylinder, it speaks in favour of a benign stenosis; likewise, a smooth tumour, occupying the pyloric section, may confirm a benign process. We must, however, be exceedingly reserved in our judgment as regards the consistence and other conditions of the tumour. Only the ensemble, the age of our patient, the long duration, the absence of a real cancer cachexia, the *real* improvements lasting for weeks and months, the absence of metastases, will confirm the diagnosis. Errors and mistakes will probably be not entirely avoided even on most careful consideration of all points in question.

Diagnosis of the Complications of the Stasis Insufficiency.

(1) *Tetany.*

A grave, though comparatively rare complication of obstructive insufficiency is *tetany.*[88] It is characterised by attacks of tonic, usually bilateral convulsions, starting from the fingers or toes and progressing centripetally. Most frequently the upper extremities are affected. As the spasm affects chiefly the *flexor muscles,* peculiar contracted positions are exhibited. The fingers begin to be flexed, the thumb is adducted, the hand hollow and flexed. Fingers and hand take hereby, as Trousseau pregnantly described it, *the position of an accoucheur's hand;* in the course of the attacks the wrist becomes flexed, somewhat rarer the forearm, and the upper arm is pressed to the body. In rarer cases the *extensor* muscles are seized also, whereby the greatest variety of convulsive positions of the muscles and joints may present themselves. On the lower limbs, the toes are considerably flexed and closely pressed to one another so that the big toe stands below the other toes and the sole of the foot becomes hollow, similar to the palm of the hand. The heel is drawn upward through contraction of the muscles of the calf, whereby the position of the pes equinus and equinovarus, less frequently that of the pes equinovalgus develops. The muscles of the femur and of the hip are only exceptionally affected. According to the extension of the convulsions, three different degrees of tetany (Trousseau) are distinguished, namely: a mild type, in which only the muscles of the extremities are seized; a medium type, when also the trunk of the body, the abdomen, and the face are involved; and finally a grave type, when the muscles of the larynx, pharynx, and tongue are implicated. Several times, and that in the fatally ending cases, increase of the temperature to 39.7° C. was observed; albumen was repeatedly found in the urine in fatal cases (tetanie albuminurique) (Imbert-Gourbeyre, Delpech, and Rabaud). Even in cases with a favourable course, there may be a temporary disturbance of the intellect, weakness of memory, disorders of speech and vision. The prognosis of gastrogenic tetany is always grave; according to Bouveret and Devic the rate of mortality is almost 70 per cent.

I had occasion to observe a favourable ending in cases of tetany in my consultation practice.

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[88] Compare Monograph of L. v. Frankl-Hochwart, Die Tetanie, Berlin, 1891, and the same writer in Nothnagel's spezieller Pathologie und Therapie, Bd. 11, 2 and 4 Abtheilung, and Bouveret et Devic, Recherches cliniques et expérimentales sur la tétanie d'origine gastrique, Revue de médecine, Janvier and Février, 1892 (there the most comprehensive bibliography on tetany complicating dilatation of the stomach).

P., 38 years of age, engineer of fire-department, suffered five years ago of gastric ulcer, followed by pyloric stenosis. Patient very much emaciated. Stubborn constipation, diminished diuresis, severe thirst. Appetite good, sometimes bulimia. Examination of stomach-contents shows presence of partly decomposed substances which, in the beginning of the treatment, smelled very much of H_2S. The amount of HCl was first increased, later it decreased; several times the contents in the morning did not contain free hydrochloric acid. On microscopic examination, always numerous sarcinæ bales; occasionally a few yeast fungi were found.

The health of the patient improved through lavage and suitable diet; he gained in weight, and was able to be temporarily without medical attention. In the autumn of 1893 he suffered from a severe attack of influenza, whereby his strength was considerably reduced. Since that time great thirst and lasting constipation. On examination of the gastric contents only temporarily food-remnants to a moderate extent were found in the morning.

In January, 1894, tetaniform symptoms appeared for the first time; his hands became convulsively cramped, simultaneously, sensation of numbness and formication in the extremities, especially the lower ones; the patient did not pay much attention to these rapidly transitory manifestations.

In the morning of March 4, 1894, after great thirst and constipation had existed for a few days just previous, suddenly severe attack of convulsions, starting from the upper extremities and later seizing the lower ones. From the extremities the attack went upward to the muscles of the shoulder and chest, so that respiration was very much laboured. The attack lasted about half an hour, and was repeated several times. Consciousness was fully retained. I saw patient at 11 A. M., after several seizures had passed. I found his hands spasmodically contracted, thumbs drawn inward, cold and exceedingly pale; face pinched; arms and legs could be moved only labouriously. Respiration retarded and painful. Patient could speak a few words incoherently. Pulse somewhat accelerated, but otherwise of good quality. He complained of burning thirst. Secretion of urine scanty; bowels constipated.

I directed the patient to be covered with hot sheets, hot-water bags to be applied to the extremities, and a litre of warm milk to be introduced into the rectum. Marked improvement followed; the spasms in the hands loosened, the extremities became warmer. After half an hour the condition was tolerable. I ordered repeated introduction of warm milk and port wine into the rectum, patient received a little beef-tea with rice-slime by the mouth. In the course of the day several slighter seizures; in the evening I found the patient weak, but in otherwise satisfactory condition. I investigated whether by pressure on the larger vessels of the forearm an attack would be produced; only a slight contraction of the flexor muscles of the hand occurred, which promptly disappeared after removing the pressure. Chvostek's phenomenon could not be produced. The following day the patient's condition, excepting some stiffness in the limbs and pain in the chest, was satisfactory, and remained so. Similar attacks did not reappear. The patient, while staying at the seashore for some time, gained considerable weight. The diuresis increased by continued use of nutrient enemata (varied between 1000 and 1200 cubic centimetres); the mechanical disturbance of the stomach was present to the same extent. On October 23, 1894, on my advice, patient was operated on; pyloroplasty performed, with perfect and lasting result.

Besides this case, I had a second case of tetany reported by my assistant, Dr. Hans Ury.[89] Trousseau's and Chvostek's phenomena were pronounced. Patient died in a short time. On post-mortem examination, besides a pyloric stenosis, caused by cicatrised ulcer, the rare occurrence of a gastric diverticle was found. Patient had suffered from a slighter attack of tetany three months before his admission to the ward.

Besides these typical attacks, there occur other tetaniform, evidently tonic convulsions in the extremities, which are associated with more or less disturbance of the consciousness. Ury described in his essay a case of this kind from my private clinic. Fleiner[90] and v. Frankl-Hochwart[91] mention similar cases.

Finally, there are certain abortive forms of tetany in cases of gastric insufficiency, which manifest themselves through creeping, numbness, formication, a sensation of peltiness; occasionally, there may develop slight tonic contractions, particularly in the hands, or even in the facial muscles. Frequently these abortive forms may continue for a long time, but in some cases they form the introduction to the classic attacks. Without doubt it seems necessary to pay close attention to those first hints, and to keep the patients under observation.

Objective symptoms of great diagnostic and differentially diagnostic importance for the diagnosis of tetany are Trousseau's, Erb's, and Chvostek's phenomena.

1. *Trousseau's Phenomenon.*—Attacks of tetany can be produced by pressure on the chief nerve-trunks and blood-vessels, so that the arterial and venous circulation is impeded. After the duration of this pressure for one or two minutes the attack commences, and ceases again after the pressure has been removed.

2. *Erb's Phenomenon.*—Erb, as the first, found an increased galvanic and faradic irritability in all motor nerves, readily accessible to examination, excepting the facial nerve. According to the most extended investigations of v. Frankl-Hochwart, the increased galvanic irritability of the nerves is to be considered as an almost constant symptom, while the faradic irritability is normal in the majority of cases and increased in the others.

3. *Chvostek's Phenomenon.*—Chvostek first elicited a mechanical super-irritability of some nerves of the extremities as well as of the facial nerve in particular. A slight tap with the percussion hammer or the finger in the course of the facial nerve will at once throw the muscles to which it goes into rapid, lightning-like contractions. If the finger is passed

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[89] Hans Ury, Deutsche medicinische Wochenschrift, 1900, No. 29 and 30.

[90] Fleiner, Archiv für Verdauungskrankheiten, 1896, Bd. 1, P. 243.

[91] v. Frankl-Hochwart, *l.c.*

with some pressure over the face downward, starting from the upper temporal section, half way between the external angle of the eye and the ear to the centre of the chin, there occur pronounced contractions in all facial branches, because evidently all branches of the facial nerves are exposed to a quick pressure by this manipulation. (Fr. Schultze.)

The tetany of gastric dilatation is a comparatively rare complication. According to Gumprecht,[92] the number of cases reported till 1897 was forty; since that time about half a dozen new cases have been added. The majority of the cases were caused by pyloric stenosis after ulcer, less frequently by stenosing pyloric carcinoma. Besides, there were observations of tetany in cases of other gastric and intestinal diseases, as, for instance, compression of the duodenum by the gall-bladder filled with gallstones (Blazicek[93]), by entozoa (Riegel[94] and others), by chronic diarrhœa (Ewald and Jacobsohn[95]), by torsion of the stomach (Fr. Müller[96] and others).

The views on the pathogenesis are divided. Kussmaul[97] was formerly inclined to refer the tetaniform convulsions to condensation and thickening of the blood, and desiccation of the nerves and muscles in consequence of violent vomiting and loss of water; recently, however, this author retroverted from his former opinion. Fr. Müller[98] considers tetany as a reflex process originating in the stomach, and bases his view upon the fact that tetany may occur in a variety of intestinal diseases with irritation of the mucosa, even in the presence of entozoa; in one of his cases he could produce an attack by mere tapping on the epigastrium.

French authors, and also some Germans, such as Loeb,[99] Bamberger,[100] Albu,[101] and others, supporters of Bouchard's doctrine on auto-intoxication, consider tetany in complication with gastric dilatation as the effect of certain toxins taken into the circulation. Bouveret and Devic tried to support this view by the proof that when the alcoholic extract of slowly dried gastric contents of patients afflicted with hyperchlorhydria was injected into a vein of a rabbit, convulsions or death were caused. Particularly, digestive mixtures with a great superabundance of free hydrochloric acid were shown to possess such toxic qualities. Cassaët and Féré,[102] however, were able to isolate the same substance from gastric contents of other persons not suffering from tetany. Fleiner[103] and Gottlieb employed gastric contents, taken immediately after a convulsive attack and manufactured after the method of Bouveret and Devic, and observed after the subcutaneous injection of the same into guinea-pigs clonic contractions, but nothing resembling tetany. Gumprecht's experiments also were without result. Finally, recent observations have shown that tetany occurs also in cases of pyloric carcinoma in which hydrochloric acid was absent, but lactic acid was abundant. It is worth while to mention that Kulneff[104] also could produce toxic bases (in one case, æthylendiamin) from the contents of dilated stomachs, with or without carcinoma. As seen, the doctrine of auto-intoxication is still without a solid scientific foundation.

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[92] Gumprecht, Centralblatt für innere Medicin, 1897, No. 24. (Excellent description of gastric tetany.)

[93] Blazicek, Wiener klinische Wochenschrift, 1894, No. 44, 46, 48.

[94] Riegel, Deutsches Archiv für klinische Medicin, Bd. 12.

[95] Ewald and Jacobsohn, Verhandlungen des 12. Kongresses für innere Medicin, 1893.

[96] Fr. Müller, Charitéannalen, 1888, Bd. 13.

[97] Kussmaul, Deutsches Archiv für klinische Medicin, 1869, Bd. 6, S. 455 etc.

[98] Fr. Müller, *l.c.*

[99] Loeb, Deutsches Archiv für klinische Medicin, Bd. 46, S. 98.

[100] Bamberger, Referiert Centralblatt für klinische Medicin, 1892, S. 1009.

[101] Albu, Volkmann's Sammlung klinischer Vorträge, 1899, No. 254.

The diagnosis of tetany is readily made under consideration of the symptoms mentioned above.

[It is but in recent times that another cause has been found for the development of that form of tetany which usually had been regarded as dependent on obstructive disease of the pylorus or obstructive dilatation, namely, the presence of foreign bodies in the stomach.

In a report in the Annals of Surgery, December, 1904, Dr. James P. Warbasse gives the history of a man who was a professional swallower of metallic substances. Warbasse mentions briefly the surgery of tetany, giving credit to Mayo Robson as the first to present the surgery of this condition. Albu, Germain Sée, and Berlozheimer have recognised the importance of mechanical gastric irritation as an ætiological factor. Warbasse was unable to find in the literature the report of any case due to foreign bodies in the stomach. Cunningham, Annals of Surgery, April, 1904, records seven cases treated surgically, and an eighth case from his own observation. Three cases were reported by Mayo Robson, two by Fleiner, one by Gumprecht, and one by Caird.[105] Warbasse's case deserves special mention, on account of the peculiar circumstances. This "human ostrich" had for several years been swallowing articles passed up from the audience. He increased the number and size of articles swallowed, but had no bad immediate attacks. He had suffered with frequent cramps in the calf of the right leg during the past two years. This cramp was often so severe as to awaken him from sound sleep. He was seized at various times with convulsions. On April 30, 1904, he swallowed nails, keys, and two watch-chains. While still on the stage he was seized with vomiting and spasm of the throat-muscles, and fell unconscious in a tetanic attack. Was removed to the German Hospital. In this, as in the previous attacks, there was a succession of spasmodic seizures. Practically all of the voluntary muscles were involved—legs, arms, neck, face, and extensors of the trunk. Often the contractions of the extensor muscles of the spine and legs were so strong as to cause the whole body to bound from the floor in a position of opisthotonos.

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[102] Cassaët et Féré, Société de Biologie, June 24, 1894.

[103] Fleiner, Archiv für Verdauungskrankheiten, 1896, Bd. 1.

[104] Kulneff, Berliner klinische Wochenschrift, 1891, No. 44.

[105] [*Vide* Boas, this book, P. 508.]

Sometimes during the intervals between attacks the patient regained consciousness, and entered into conversation; at other times the spasms followed in quick succession. These seizures presented three stages: (1) All of the attacks were preceded by vomiting or a violent effort at vomiting. (2) This was always followed by a sensation of choking, as though he were being strangled and could not get air—evidently as spasms of the glottis muscles. (3) He then fell unconscious to the floor, and the convulsions immediately supervened. During the first two stages there was always a sensation of dizziness. After from one to six or eight convulsions the attack subsided, and the patient went about his business.

Fluoroscopic examination and an x-ray picture showed a mass of foreign matter in the region of the cardiac end of the stomach. This mass was in the form of a ball or nest; no separate foreign substances could be detected in any other location.

Operation, May 3d. Abdomen opened through left rectus muscle, and stomach brought into wound. Transverse opening made in stomach over cardiac end. The hand, introduced in this opening, discovered a mass of metallic substances felted together and bound by chains into a nest. This was separated, and the following articles removed: seven pocket-knives, seven door keys, twenty nails (two and one-half inches long), one small spoon, one button-hook, an ordinary pin, a knife-spring, and two watch-chains (one gold-plated, the other silver). The total weight of these things was sixteen ounces (480 grammes). The mucous membrane of the stomach appeared slightly congested, but otherwise normal. There was some dilatation of the left end of the stomach, induced by the weight of its contents and the ineffectual efforts at expulsion. There were no peritoneal adhesions or evidences of irritation, excepting the adhesion of the stomach to the former scar, which was about in the middle of the organ, and which had encouraged the development of a cardiac pouch. (Patient had been operated on once before, on January 3, 1901, at St. John's Hospital for the same trouble. A gastrotomy had been done, and the following articles had been removed: one hundred and twenty-nine ordinary pins, two horse-shoe nails, twelve wire nails, two keys, and two watch-chains. At that time he made a good recovery, and was discharged three weeks later.) The stomach-wound was closed with chromic gut sutures, and the slack of the stomach-wall taken up about two inches by making a longitudinal fold, approximating two surfaces about an inch broad. The patient was kept on nutrient enema for five days, being allowed a small amount of water by mouth from the first. At the end of five days he was permitted fluid diet, and two weeks after the operation full diet. He was discharged from the hospital, cured, at the end of three weeks.

The patient recognised one knife that he had swallowed five months before. The button-hook had been swallowed ten months before.

The symptoms of tetany in this case were not recognised until after the operation, when a history of his recent attacks was obtained, and the facts concerning previous attacks inquired into. The symptoms described by Trousseau, Chvostek, Erb, and Hofmann were not elicited. This man, as was the case with the patient reported by Cunningham, had repeatedly fallen in tetanic convulsions, and been in three different hospitals, and the disease failed of recognition.

Dr. Warbasse concludes with the following words: "The cause of this man's symptoms cannot be found in pyloric stenosis. What moderate dilatation he had was due to the dragging down by the weight of foreign matter, and to the efforts of the stomach to empty itself of a mass that could not pass through the pylorus. As a matter of fact, a normal pylorus may be the cause of dilatation if it is not large enough to accommodate the contents which the stomach is trying to extrude. As far as the effect on the stomach-wall goes, it amounts to the same thing as stenosis of the pylorus. It seems to me that this case speaks for the mechanical theory of the ætiology of gastric tetany.]

(2) *Gastric Hæmorrhage in the Course of Obstruction Insufficiency.*

In certain forms of obstruction insufficiency severe intercurrent hæmorrhages were frequently observed, particularly under two circumstances: first, in cases of benign pyloric stenosis; and second, in those of pyloric carcinomatous ulcer. It is true, bleedings also occur in pyloric carcinomata without ulcer, but usually never to such an extent as in the said forms. The bleedings occur especially in cases of grave benign stenosis of the pylorus; in one case I observed it also in a so-called relative stenosis. Usually, the pyloric stenosis was preceded for some time by symptoms of ulcer with hæmatemesis. At the acme of the development, frequently after dietetic errors, there occur in connection with more or less violent gastralgia severe hæmorrhages, which, as a rule, ceasing with suitable measures, may repeat themselves after a longer or shorter interval. Whether we have to deal in such cases with recrudescence of the ulcer or with corrosion of the mucosa and blood-vessels is difficult to decide. The interesting researches on rabbits by Talma[106] teach that besides chemically different gastric contents, *i.e.*, superacidity, an increased tension of the gastric wall may produce gastromalacic processes in the mucous membrane. Probably there exist similar conditions in pyloric stenosis.

Differential Diagnosis.

The differential diagnosis in stasis insufficiency must be taken under consideration, first, to demarcate the grave motor disturbance from other gastric diseases, then, after ascertained diagnosis, to contemplate the various ætiological possibilities. As the latter has already been done in discussing the various types of insufficiency, we need not revert to it again.

As to differential diagnosis of stasis insufficiency from other gastric affections, diseases such as gastritis, nervous dyspepsia, and ulcer may hardly be considered at the present time, if examination is carefully made; but the (1) physiologic large stomach (megalogastria), (2) myasthenia, and (3) gastroptosis will have to be considered in differential diagnosis.

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[106] Talma, Zeitschrift für klinische Medicin, Bd. 27, Heft 1 and 2.

1. *Megalogastria.*—We have discussed the differential symptoms (p. 483).

2. The description of mechanical insufficiency of the first and second degrees has given us the chief differential signs of either condition. It remains for us to compile briefly the diagnostic characteristics which speak for stasis insufficiency and against atony.

The stasis insufficiency is a grave disease, rarely curable by medicines; simple atony, however, is a benign affection, capable of repairs, and not essentially debilitating to the general organism, especially if suitable measures are applied. The stasis insufficiency is characterised by the abnormal accumulation of products of decomposition of the chyme in the stomach at a time when the same under normal conditions is usually free of food-remnants, as, for instance, in the morning before partaking of food. The stomach-contents show, according to the inciting cause, varied characteristics; but there is always an abnormal acid formation—either abundant hydrochloric acid secretion, or, much rarer, excessive lactic and fatty acid production. These abnormal conditions are very often associated with the pressure of sarcinæ and yeast in large quantities. In atony of the stomach, the organ is in the morning either entirely empty, or may contain a greater or lesser amount of gastric juice, and occasionally also a small amount of food remnants, which, however, do not show any signs of abnormal decomposition. Once in a while there may occur sarcinæ or yeast fungi; but in such cases they are not found constantly, nor in considerable quantities.

In stasis insufficiency caused by stenosis, at an early stage gastric stiffening and even peristaltic unrest manifests itself, but never in simple myasthenia. Diuresis in cases of stasis insufficiency is always more or less diminished, and in consequence the specific gravity of the urine is higher than normal; in gastric atony, diuresis is usually sufficient or reduced but slightly, and the specific gravity of the urine is lower.

The bowels are usually very much constipated in stasis insufficiency; such may be the case also in atony, but there are frequent cases of normal bowel movements. In the former there are occasionally the symptoms of tetany, while in atony such disturbances do not occur.

All other manifestations, such as vomiting, pain, gas formation in stomach and intestines, emaciation, sensation of cold in the extremities are found so frequently in common in either of the affections, that differential diagnosis between them could not be made, based upon these symptoms alone.

3. On the differential diagnosis between ectasia and gastroptosis, see page 545.

Treatment of Motor Insufficiency of the Second Degree.

The treatment must consider first the ætiology, and second the symptoms. The better informed we are about the inciting cause of the stasis insufficiency, the better-devised and better-aimed the treatment will be. There are numerous cases of apparently grave insufficiency which are cured completely on administration of rational internal treatment; there are other cases in which the internal treatment may act palliatively, but only the knife leads to a lasting cure. To the former type belong the spastic pyloric stenosis, which may often be removed permanently, and the atonic dilatations; to the latter type, the organic stenosis, whatever the ætiology may be. [Even in the latter cases it is worth while to make a trial with thiosinamine; in some cases a cure resulted, which has lasted for over eighteen months.] I mention first two examples of perfectly cured stasis insufficiency, one case of pylorospasm, another of severe atony.

CASE I.—H., merchant, 37 years of age. Excepting diphtheria, patient always healthy. Nothing abnormal as to inheritance. The gastric complaint has been existing for five or six years, and manifested itself first in temporary gastric pains. Patient did not pay much attention to it, ate and drank *ad libitum*, excesses in alcohol and tobacco. Gradually considerable deterioration. Regularly and soon after ingestion of food severe stomach-ache appeared, ending sometimes in vomiting. Nevertheless, patient continued his former method of life, ate and drank as in healthy days. The catastrophe came in April, 1900. The pain exacerbated to intolerableness; vomiting occurred regularly even after small meals. The vomited material consisted of copious fluid substances to a much larger extent than corresponding to the quantity of the ingesta, and had a foul smell. Eructation, too, had frequently the smell of rotten eggs. Simultaneously, complete anorexia, increased thirst, severe constipation. Patient had lost twenty-five pounds in weight. Physical examination shows dilatation of the stomach, pronounced succussion sound, food-remnants amounting to more than one litre after test-supper. In the fermenting gastric contents abundant hydrochloric acid; in the sediment sarcinæ, yeast, fatty acid crystals, abundant starch, a few muscle-fibres.

Treatment: Admittance to a sanatorium for gastric diseases, with the necessary dietetic and other directions. The result was excellent. All complaints, excepting an occasional heartburn, ceased; patient gained ten pounds in weight, and can now stand very well the food, selected, to be sure, for the purpose. The repeatedly-made examination of the stomach, before partaking of food, shows that it is empty.

CASE II.—J. H., 13 years of age, suffered for the last three years from loss of appetite and vomiting of copious sour decomposed food-remnants, repeated sometimes daily or every second or third day. Frequent overloading with indigestible food was stated as ætiological factor. The examination shows large dilatation of the stomach, no tenderness or pain. In the stomach, which was explored for almost a year, there were regularly in the morning stagnant masses, varying in amount from $^{3}/_{10}$ to $1^{1}/_{2}$ litres. Gastric contents intensely acid: HCl amounts to 0.15 to 0.19 per cent. No lactic acid. The well-known three strata; gas fermentation. In the sediment constantly sarcinæ and yeast. Diagnosis: atonic ectasy. Regular lavage in the morning is of little effect as to retention of food, because patient does not comply with the dietetic directions. After one year's treatment, relatives of the patient are advised to have the patient operated on for gastro-enterostomy, because patient had remained very backward in nutrition and development. Operation refused. Six months later patient returns well-nourished, stating that he can eat any kind of food without vomiting, and that lavage of the stomach has not been necessary for several months. Examination of the stomach in the morning exhibits but very small quantity of remnants of slightly acid reaction.

Internal treatment must, whatever the ætiology of the insufficiency may be, keep in view the same principles, namely, the removal of the impediments and the strengthening of the gastric muscles.

Diet is the most important factor as regards the former. We have hereby to consider two tasks: first, that of improving the general nutrition of the patient, who, as a rule, is very much debilitated; second, that of arresting the unfavourable motor and absorbent function of the gastro-enteric canal and the subsequent altered digestive conditions for the total alimentary apparatus. We shall approach nearest to the former problem by advising the patient to take frequent but small meals, whereby we simultaneously make favourable conditions for the propulsion of the ingesta into the intestine. The conception of "small meals" must vary for each case of insufficiency according to its degree. [Some writers have recommended two large meals, to be taken morning and evening, after previous lavage of the stomach (Bettmann).]

The particular selection of food also depends, above all, upon the degree of dilatation and the type and extent of the fermentative processes; but one thing can be said without doubt, that frequent and small meals, and among them especially solid food, can be best borne. It is only doubtful whether, as taught by van Swieten, and after him even at the present time experienced authors (among others Ewald), fluids should be forbidden entirely or almost entirely, or whether, as Liebermeister,[107] Fleiner,[108] Riegel,[109] Penzoldt,[110] Albu,[111] Pick,[112] and others advise, fluid or pulpy food should be recommended. According to my conception, the selection of food depends, as emphasised also by Riegel, not a little upon the condition of secretion. In benign pyloric stenosis with hyperchlorhydria, solid proteid substances also are very soon liquefied in cases of stenosing gastritis and pyloric carcinoma; with achlorhydria, such is the case to a much smaller extent. In the former cases, therefore, solid substances are adaptable; in the latter, as far as it pertains to solid proteid substances, they ought to be taken only in predigested condition. It must, however, be admitted that fluids in small separate quantities are well borne, even in insufficiency with absence of HCl, and are propelled through the pylorus. Riegel advises, in cases of the latter type, caution in administering carbohydrates, but this caution is valid only for the insoluble carbohydrates. Fats, at least in large quantities, are not well borne in all cases of stasis insufficiency. Very appropriate is, in general, milk in small doses, the nourishing value of which may be enhanced by addition of finely divided amylum substances (legumens, rice and oatmeal flour, mondamin, etc.).

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[107] Liebermeister, Die Krankheiten der Unterleibsorgane, Leipzig, 1894.

[108] Fleiner, Volkmann's Sammlung klinischer Vorträge.

[109] Riegel, v. Leyden's Handbuch der Ernährungstherapie, Bd. 2, P. 43 *et seq.*

[110] Penzoldt, in Penzoldt-Stintzing's Handbuch der speciellen Therapie, 2 Aufl., Bd. 4.

[111] Albu, Deutsche medicinische Wochenschrift, 1900, No. 11.

[112] Pick, Vorlesungen über Magen- und Darmkrankheiten, Wien, 1895, I Theil.

In grave cases of stasis insufficiency we seldom succeed in covering the want of fluid of the organism by the way of the stomach. The question of rectal introduction of water enters into the foreground. The diuresis must give us a kind of a standard. In cases where the urine amounts to less than 600 cubic centimetres during twenty-four hours I administer under all circumstances rectal enema, and that so frequently as necessary during the twenty-four hours till the diuresis of one litre is reached.

In cases of pronounced development of the symptoms, particularly of the vomiting and pain, and the bodily strength permitting, the patient may be fed exclusively by the rectum for a longer time (one to two weeks). In several cases of gastric ectasia after ulcer, I had the great fortune through this method to observe that the patients felt considerably better for many months than ever before. Rössler[113] and Schlesinger[114] also gained remarkable results by rectal feeding in several cases.

As to special diet, the following principles have to be considered. The quantity of beverages allowed may amount to about 1000 to 1500 cubic centimetres during twenty-four hours, and when the motor function is especially reduced, to even less. The fluids to be taken should be administered with the view of nutritiousness—above all, as already mentioned, milk and particularly cream, the digestibility of which may be enhanced by addition of some brandy. Tea, even coffee, in small quantities, may be allowed. Among other fluids, beef-tea, yolk of an egg, gruel in doses of 50 to 100 cubic centimetres may be recommended. Acidulous mineral waters, as well as all other mineral waters, are to be limited within the above-mentioned amount. [In the administration of mineral waters the patients are often sinned against, even by physicians of great renown, as I had the experience while spending a season at Bedford Springs, Pa.

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[113] Rössler, Wiener klinische Wochenschrift, 1893, No. 40.

[114] Schlesinger, *ibid.*, 1895, No. 19.

The indiscriminate advice of physicians to go to watering places and to drink large quantities of mineral waters without having made a careful examination of the condition of the stomach, is certainly a wrong to the patients.] If great thirst is present, ice, iced milk, iced coffee, and iced tea are very appropriate substances, but it will be advisable to caution the patient against the dangers of a precipitate quenching of the thirst. To mention it here, Riegel recommends Dover's powder (0.4) as a thirst-quenching remedy.

As to rectal feeding, the authorities do not agree on details, but, as a whole, emulsified soft-boiled eggs (consistency of a plum) or peptone, bouillon, claret, form the chief matter (see page 304). The nutrient enemata, applied by myself, and well tried, have been described (page 455). Such nutrient enemata may be administered two or three times during the daytime.

In the selection of the food the amount of water contained in it must be considered, too; on account of this, roasted meats are to be preferred to the boiled ones, rich in water. Furthermore, the bread (wheat and rye bread) is to be cut in slices and toasted, or only the crusts are allowed. Likewise vegetables rich in water, as well as fruit conspicuous for its amount of water, ought to be avoided.

Among the fats, fresh butter and cocoanut butter, which is almost free of fatty acid, may be permitted in small doses,* but in such cases, too, it depends upon the subjective tolerance and the result of the examination of the gastric contents.

A remedy of considerable palliative value is lavage of the stomach. Its therapeutic importance rests upon the removal of stagnant chyme remnants; in cases where such an abnormal accumulation does not exist, or no longer exists, gastric lavage is superfluous.

Gastric lavage is effected at the present time almost everywhere by means of a siphon apparatus; the stomach-pump, not offering any advantage, has been rightly forsaken. Lavage may be performed with warm water, or with addition of various kinds. In cases of pronounced hyperchlorhydria and sarcinæ and yeast fermentation, I usually prefer to use an addition of sodium bicarbonate (1 to 2 per cent.); in lactic and butyric acid fermentation, as well as fœtid decomposition, antiseptic additions may be made, such as, for instance, resorcin (2 to 3 per cent.), sodium salicylate (1 to 5 per cent.), salicylic acid (3 to 1000), boric acid (3 per cent.), sodium benzoate (1 to 3 per cent.), chloroform water (1 per cent.), creolin (10 to 15 drops to 1 litre), or better, lysol in the same dose; finally, ichthyol (10 to 20 drops to 1 litre of water). The latter has proved itself efficient, particularly against fœtid decomposition, in a few cases. A really eclatant and lasting usefulness of any of these antiseptic additions, however, was not observed by me; especially, I never succeeded in removing sarcinæ from the gastric contents by means of these antiseptics, unless by strengthening of the gastric muscles the stagnation itself disappeared.

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* Who ever has frequently removed the contents from a severely dilated stomach in the early morning will know that the same frequently contains large quantities of fat.

The question at what time the stomach should be washed out has been discussed in the General Part (page 349). I only emphasise that the most favourable time is in the morning, because the stagnant contents of the jejune stomach are unfit for absorption and propulsion into the intestine. In the evening I use lavage only if severe pain, nausea, and inclination to vomiting demand it. In severe cases it is occasionally necessary to rid the stomach of stagnant masses in the morning and evening.

The usefulness of gastric lavage manifests itself, first, through the subjectively improved condition, the increase of appetite, the ceasing of pain; objectively through the ceasing of vomiting, increased diuresis, lessening of the constipation, gain in body weight. The quicker the effect of the gastric lavage ensues, the more rapidly the stagnant masses decrease, the better the prognosis. If by lavage little or nothing at all is gained, the prognosis is always triste.

In favourable cases and under the effect of the gastric lavage, a mechanical insufficiency of the second degree frequently becomes one of the first degree; and instead of stagnant contents in the morning, only gastric juice is found. As an instance of this kind, I shall discuss a case in the section on gastro-succorrhœa.

Partly on account of occasional errors in diet, but especially on account of the surely increasing disorders in compensation, particularly in pyloric stenosis, the effect of lavage, as already mentioned by v. Leube and Oser for a long time, gradually decreases and finally ceases entirely. At this stage the patient feels most wretched, and vomits after the ingestion of the smallest quantity of solid or liquid food. In other cases the reflex excitability has become so slight that not even vomiting occurs, but on corresponding filling up of the stomach there is an "overflow." Usually, the effect of the lavage, as well as of the general treatment, depends on the inciting cause. In carcinomatous pyloric stenosis, even before an advanced stage, the effect of the treatment will evidently always be less satisfactory than in cicatrised or spastic stenosis or atonic ectasy.

In very debilitated patients I replace gastric lavage by simple expression, which, however, has not the same effect as the former, but can be performed in a much shorter time. I advise patients with stasis insufficiency, who on account of overfilling of the stomach are disturbed in their night rest, to get rid of a portion of the gastric contents by expression in the evening before going to bed. The effect of this procedure is not essentially inferior to the thorough washing out of the stomach.

A new procedure, recommended by P. Cohnheim[115] for the cure of spastic and organic stenosis, namely, the oil method, deserves to be mentioned. For some time 50 cubic centimetres of pure olive-oil is to be taken or introduced by the tube, three times a day, one hour before meals, or once a day 150 cubic centimetres of olive-oil warmed to body temperature is introduced into the empty stomach. According to Cohnheim, the administration of oil corresponds to three indications, namely, as an antispasmodic, diminution of friction, and improvement of the state of nutrition. To this may be added the observation made by Strauss, Mathieu, and others, that the fats considerably diminish the production of hydrochloric acid. Cohnheim recommends the said procedure particularly in spastic stenosis, but also in organic stenosis, whereby he observed considerable improvement or even cure. Further observations will have to demonstrate the applicability, the value, and the limitations of the method.

Next to gastric lavage, the external and internal faradisation of the gastro-intestinal region is the most frequently applied and best approved remedial agent. The technique has already been briefly described (page 487; see also page 334). An effect from faradisation might be expected perhaps in cases of muscular atony, while in pyloric stenosis the gastric muscles have spontaneously become so hypertrophic that further aid will be hardly necessary, and can hardly be further expected.

Gastric massage, connected usually with massage of the whole abdomen, may pursue two aims: first, to propel the stagnant chyme into the intestine; or second, to better nourish the gastro-enteric muscles by enhanced blood-circulation, and to strengthen them for better compensation. The first purpose is not without the danger of transplanting the numberless infectious germs of the half putrid chyme into the intestine. Moreover, it is quite undecided whether we really can succeed in propelling by massage the greater portion of the contents through the pylorus. In pyloric stenosis the effect of massage must not be estimated very highly, but better prospects might be had in myasthenia, though in the latter cases also we must not expect too much.

The hydrotherapeutic measures also are for the purpose of increasing the appetite, of improving the general state of nutrition, and of influencing the local process. Hydropathic packs (Umschläge) and douches to the gastro-enteric region, and friction, are nevertheless good aids in the treatment of ectasy of the stomach.

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[115] P. Cohnheim, Archiv für Verdauungskrankheiten, 1899, Bd. 5, Heft 4; Deutsche medicinische Wochenschrift, 1900, Vereinsbeilage No. 6; 13 internationaler med. Kongress in Paris, reported in Berliner klin. Wochenschrift, 1900, P. 740.

Because, as is well known by experience, the adipose tissue disappears rather rapidly, the use of well-fitting abdominal bandages is to be recommended as an appropriate palliative remedy. The number is so large that it becomes difficult to discuss even a part of the recommended supporters. I prefer Bardenhener's abdominal corset (see page 343), or a rubber bandage made of a single piece fastened to a pelvic belt and closed anteriorly by a belt.

The *medicinal treatment* is intended to increase the motor function of the organ and to decrease the processes of decomposition in the stomach (gastro-enteric antisepsis). In order to stimulate the motor function, the strychnia preparations (extractum nucis vomicæ, 0.03 to 0.05, or also strychnia nitrate, 0.01, hypodermically) have frequently been employed. Their effect in cases of pyloric stenosis is, for the above-given reasons, very doubtful.

Kuhn[116] recently, experimenting with a greater series of anti-fermentative remedies in regard to their capability of inhibiting gas formation, came to the conclusion that salicylic acid, for some time recommended as preventing gastric fermentation, in doses of 0.5 to 1.5 and sodium salicylate (1.0 to 2.0), also saccharine and sodium benzoate (0.5 to 1.0 to 2.0) are in the front rank. In my practice, sodium salicylate in doses of 1.0 to 2.0 grammes *pro die* has often been of great service.

In cases of marked lactic and butyric acid fermentation a persistent administration of hydrochloric acid (10 to 15 drops in water) has been of some use. Naunyn first recommended large doses of carbolic acid (0.1 to 0.2)—for instance:—

℞ Acidi carbolici	2.5
Pulveris radicis althææ	q. s.

u. f. pilulæ No. xxv.
Sig.: One pill three times a day.

But I did not observe any special effect.

Other drugs to be mentioned are bismuth salicylate, salol, beta-naphthol, benzonaphthol (Ewald),[117] and menthol. The French, particularly (Bouchard, Dujardin-Beaumetz, and others), are great eulogists of the anti-fermentative treatment of gastric diseases. A real effect is not to be expected from any other of these drugs. Their use contributes only to the quieting of an immoderately developed pharmacological conscience.

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[116] Kuhn, *l.c.*

[117] Ewald, Berliner klinische Wochenschrift, 1892, No. 26 and 27.

H. Menche[118] highly values resorcin for various gastric affections, including ectasia, and recommends resorcin in solution:—

℞ Resorcini resublimati (Merck)	2.0 : 175.0
Tincturæ rhei vinosæ	5.0
Syrupi simplicis	20.0

M. D. in vitro nigro.
Sig.: One tablespoonful twice a day.

I observed occasional results, and usually give it dissolved in chloroform water:—

℞ Resorcini resublimati	2.0
Aquæ chloroformii saturatæ	150.0

Sig.: One tablespoonful three times a day.

A few symptoms sometimes require special treatment, for instance, vomiting, anorexia, insomnia, and constipation.

The best measure against vomiting in stasis insufficiency is, as emphasised above, gastric lavage. If we do not succeed with lavage, we will not succeed with medicine. In such cases feeding by the mouth must be limited to the smallest measure possible, while rectal feeding must be applied to a large extent.

Against anorexia, bark of condurango has been used very frequently, best in form of fluid extract, in doses of 1 teaspoonful in water three or four times a day. It may be appropriately combined with fluid extract of colombo, or with creasote or extract of nux vomica.

If sleeplessness is present as result of pain, the above-mentioned narcotics may be considered; in nervous insomnia, sulphonal or trional (1.0 dissolved in boiling water and cooled to a temperature of 40° C.), or chloral hydrate in suppositories (2.0), may be administered.

As a rule, I do not prescribe purgatives for constipation, but I regard irrigation with soap-suds, castor or cod-liver oil emulsion, glycerine, etc., as sufficient. As the diet is an exceedingly bland one, daily evacuation need not be insisted upon, unless disturbing symptoms (headache, marked flatulency) demand it.

No definite principle has been laid out for the treatment of tetany; as a rule, narcotic or stimulating remedies must be resorted to. For the former, I would recommend the rectal administration of chloral hydrate (2.0 to 3.0 pro dosi) as the best. Of not less importance is the promotion of diuresis, whereby the toxins circulating in the blood are eliminated quickest. In the case described above, repeated introduction of milk into the rectum proved apparently very efficient. In another case, though less severe, this measure was also helpful.

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[118] H. Menche, Centralblatt für klinische Medicin, 1891, P. 377.

Mineral waters are decidedly contra-indicated in cases of stasis insufficiency, likewise the systematic use of commercial sour waters; but there is no objection to the use of moor (mud) baths, salt-baths, and chalybeate baths. For the after-treatment of stasis insufficiency, either improved or cured by surgical intervention, an abode at the shore or in the mountains is to be recommended. Those resorts deserve preference in which there is the possibility of a rational diet.

Surgical Treatment.

Almost simultaneously with the introduction of the radical operation for malignant tumours of the stomach by Billroth and his disciples, there developed the endeavour to restore functional activity to the dilated stomach by means of surgical intervention. It deserves to be mentioned that Kussmaul,[119] in 1869, foresaw the possibility of surgical treatment of pyloric stenosis. It is a command of piety toward the Pastmaster of the Clinics, to reiterate here his memorable words, written nearly forty years ago: *"Whether perhaps bolder generations of a far distant future will undertake to obtain in such cases radical results through gastrotomy, formation of a gastric fistula, and divulsion of the stricture by knife or bougie, who would venture to answer at the present time? One must even fear of reaping still or loud mockery by merely asking the question."*

There is no uniformity of opinion among surgeons as to pertinent methods of operation; indications for the individual method become more distinct and precise, the larger experience grows. Not a little in this regard has been contributed by the perfected methods of examination, and this fact was repeatedly admitted by the surgeons; for the further development of the surgical treatment of gastric insufficiency, the greater preciseness and subtilisation of the diagnosis will be of greatest importance.

The advice for surgical procedure in cases of gastric insufficiency depends first upon the answer to the preliminary question as to the ætiology of the affection, and above all, whether the process is malignant. In the latter case surgical intervention may serve under favourable conditions as life-prolonging but not life-preserving. If a malignant process can be excluded, the more exact knowledge of the cause of the mechanical insufficiency, which in many cases at least may be elicited with certainty, is desirable, of course, but not at all necessary.

In discussing a surgical intervention, the following questions must be considered:—

1. Is an operation necessary?

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[119] **Kussmaul, Deutsches Archiv für klinische Medicin, 1869, Bd. 6, P. 485.**

2. Does the operation still offer any chance in the condition in which the patient is existing?

3. Which method should be selected?

The first question is not at all easy to answer. If a more extended experience confirms the above (p. 515) reported observations that stasis insufficiency in apparently grave cases of long standing is frequently cured, we cannot haphazard recommend the patient to undergo the risk of an operation, which may be not without peril. As a whole, it will be to some advantage, especially in cases of pylorus spasm, which may be favourably influenced by internal treatment, to limit oneself to long-continued palliative measures (oil-cure, lavage, diet, etc.). It cannot be denied that sometimes certain complicating circumstances, such as frequently repeated hæmorrhages or recrudescences, may give occasion for an operation; the same must, however, in this purely transitory form, not be recognised as a generally valid operative measure. The same is the case with insufficiency dependent upon atony. In these cases, also, surgical treatment will be the exception. The palliative measures must be applied thoroughly at least for a long period of time before we appeal to the surgeon. It is different as to organic stenosis. In these cases also there may not be a fully developed pyloric stenosis; the hypertrophic muscles may do their function for a long time and prevent disturbances in compensation. But in the majority of cases which come under our observation, the process has progressed so far that a real and lasting cure, or even improvement, can be gained usually only by means of the knife. The indication for an operation is, according to the circumstances of the individual case, more or less urgent. A good standard criterion for the gravity of the case rests in the careful measuring of the retained contents. If the same, under appropriate treatment, becomes gradually lessened, or if perhaps very soon gastro-chylorrhœa occurs instead of gastric stagnation, or if the stomach, after a few washings-out, is empty in the morning, the prognosis may usually be considered good. Cases of this kind do not belong to the surgical forum. Besides the stagnant masses, the general strength of the body, the weight and diuresis must require our attention. Putting these three factors together we may very soon come to a decision as to what prospects we may have in the internal treatment of the case in question. Furthermore, the social condition of the patient must be considered. I would, for instance, not, or only exceptionally, recommended the operation to an official, living in quiet circumstances, but I would recommend it to a saloon-keeper or wine-agent, in whom a good stomach is an absolute necessity. Fleiner[120] states quite correctly that the pecuniary circumstances may also have to be taken into consideration.

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[120] Fleiner, Volkmann's Sammlung klin. Vorträge, 1894, N. F., No. 103.

For instance, in the case of a labourer who must provide daily bread for a large family, and cannot provide the prescribed diet for himself, an operation may become necessary at an earlier time than in case of a well-to-do woman, who can afford the luxury of an appropriate diet.

The second question, as to whether the operation offers a good chance under the condition of the reduced nutrition and at the stage of the process, must likewise be carefully discussed. In general, the majority of patients decide for operation only at a period when they themselves are convinced of the uselessness of any and all palliative measures. This period often coincides with that of extreme inanition. It is evident that under such circumstances any good effect of the operation can hardly be expected; and yet even in such apparently desperate cases favourable results have been observed. Even after an attack of tetany, operative cures were observed by Albu and myself (p. 508).

The third question pertains to the choice of the operative method. Under certain circumstances, namely, if the diagnosis is clear, the operative method and even the prognosis may be decided upon from the onset. In other less clear cases, the choice of operative method must be left to the surgeon, according to the conditions found during the operation.

(1) *Loreta's digital divulsion of the pylorus.*—The nature of the method consists of exposing and opening the stomach, after previously careful lavage, by an incision parallel to the costal arch, and subsequent expansion of the pylorus by introduction of one or both index fingers.

As to the results of the operation, Haberkant[121] has compiled 31 cases, with 19 cures and 12 deaths—a mortality of 38.7 per cent.

The drawbacks of the operation, not frequently performed in Germany, are the following: the digital divulsion does not prevent return of the stenosis; in several cases the operation had to be repeated. Furthermore, cases have been reported in which the digital divulsion was attempted, but did not succeed. In such a case Novaro[122] had to make resection of the pylorus; in another, pyloroplasty; in the latter case the mucosa was so much torn in two places by the attempts at dilatation, which had no effect, that the patient died. Furthermore, the proceeding may fail on account of marked thickening and callosities of the pyloric wall, or of adhesions of the pylorus with neighbouring organs. Loreta's digital divulsion is accordingly an insecure procedure, not entirely without danger, and applicable only in a certain number of cases of pyloric stenosis. Terrier and Hartmann,[123] therefore, correctly state: "C'est une opération a abandonner et qui ne doit plus rentrer que dans le domaine de l'historie."

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[121] Haberkant, Archiv für klin. Chirurgie, 1896, Bd. 51, P. 535.

[122] Novaro, Contrib. alla chirurg. dello stomaco, Sienna, 1890.

[123] Terrier and Hartmann, *l.c.*, P. 326.

2. *Tucking the Gastric Walls According to Bircher*[124] (*Gastroplicatio*).—This method is applicable exclusively in cases of atonic dilatation. The above-mentioned author conceived the possibility of increasing the motor activity of the stomach by lessening the size of the gastric cavity; this he sought to perform by establishing folds in the gastric walls. Thus the greater curvature is elevated; that is, the lowermost point of the stomach is raised to such a position that the contractions of that organ are able to force the ingesta through the pylorus. The operation consists in making an incision 15 centimetres in length down to the peritoneum, which is then also incised. Next the stomach is lifted out of the wound and spread out flat; for this purpose the edges of the wound must be markedly retracted. The anterior wall of the stomach is then made shorter by means of a longitudinal fold; in securing the latter the two extremities of the greater curvature are united by means of a suture with the corresponding points of the lesser curvature, after which the fold between these points is also sutured. Thus a fold of tissue is suspended within the stomach, which runs parallel to the longitudinal axis from above downward. Bircher obtained favourable results in three cases (one died three months after operation, from cancer of the pylorus). Bircher favours diminishing the size of the stomach by means of an operative procedure whenever medication and mechanical therapy give but temporary relief. Aside from Bircher himself, contributions have been made on the subject of gastroplication by Weir, Brandt, Ewart and Bennett, Boretius Nicholaysen, Shattock, Porter and Wright, Roux, Moynihan, Tricomi, Hartmann.[125] Surgeons all agree that this procedure need rarely be considered in cases of atonic insufficiency, for equally good results are obtained by means of suitable diet and gastric lavage. It is also quite evident, as has been very properly stated by Ullmann,[126] that Bircher's method has influence upon the size but not upon the relaxed condition of the organ. This operation has been performed only rarely.

3. *Heineke-Mikulicz Pyloroplasty.*—This operation, which has been devised independently by v. Heineke[127] and by v. Mikulicz,[128] consists, after opening the abdominal cavity and drawing the stomach up, in incising the pylorus and, if constricted, in splitting it in its longitudinal axis. The longitudinal wound, which extends right and left into the healthy wall of

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[124] Heinrich Bircher, Korrespondenzblatt für schweizer Aerzte, 1891, No. 23, and 1894, No. 18.

[125] Quoted from Terrier and Hartmann, *l.c.*, S. 339.

[126] Ullmann, Münchener medicinische Wochenschrift, 1895, No. 19.

[127] v. Heineke, Fronmüller, Operation der Pylorusstenose, Inaug.-Diss., Fürth, 1886.

[128] v. Mikulicz, Verhandlungen des 16 Kongresses der deutschen Gesellschaft für Chirurgie, 1887.

the stomach and duodenum, is converted into a vertical one by retracting the edges of the wound at the middle; this vertical wound is then closed by means of several rows of sutures. The posterior wall of the new canal is then formed by the old scar and the anterior wall by the healthy portion of the duodenum and the stomach. The results of pyloroplasty are quite favourable, according to the recent comparisons of Canjole, Terrier and Hartmann. Of 121 cases of pyloroplasty only 19 died; that is, a mortality of 15.7 per cent. Pyloroplasty is as little likely to prevent recurrence as gastro-enterostomy, which is to be described later; this fact may be attributed to a certain extent to faulty indications. When performed under favourable circumstances, pyloroplasty is the ideal operation in benign pyloric stenosis, even though the majority of surgeons do not value it as highly as gastro-enterostomy. According to v. Mikulicz and Kausch,[129] pyloroplasty is contra-indicated in the following cases:—

(*a*) In fixation of the pylorus, especially when the fixation is in a high position; this operation cannot prevent kinking due to fixation, and may indeed favour it.

(*b*) Pyloroplasty cannot be employed when the pylorus is infiltrated, as is the case not infrequently in ulcer and old scars, or when involved by large ulcers in its neighbourhood. The suture cannot be depended upon in infiltrated or markedly scarred tissue; furthermore, the pylorus which has been dilated by means of such a plastic operation may again become constricted as a result of further contraction of the scar tissue. A fresh ulcer in itself is not a contra-indication to pyloroplasty.

(*c*) It is always possible when marked gastric atony and ectasia exist, that the weak motor activity of the stomach is unable to force food out of the dilated pylorus, since the latter is always decidedly higher than the most dependent portion of the ectatic stomach.

[Mayo* states his experience as to pyloroplasty briefly as follows: "The pyloroplasty of Heineke-Mikulicz, in our experience, has but little risk in suitable cases, but is open to objection. It enlarges the calibre as much in an upward direction as downward in the line of drainage, and the extent to which this enlargement can be carried out is limited. The pylorus, following this operation, is exceedingly prone to become adherent, so that the opening remains at a high level. The stomach, if greatly dilated, must elevate the food to the high-lying outlet, and it frequently happens that the degenerated muscle fibres are incapable of the muscular effort, and, as a result, the patient is not materially benefited. *In three cases we fastened*

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* [William J. Mayo, The Journal of The American Medical Association, June 11, 1904; Annals of Surgery, Philadelphia, November, 1905.]

[129] Handbuch der praktischen Chirurgie von v. Bergmann, v. Bruns und v. Mikulicz, 1900, Separatabdruck, P. 23.

the pylorus, after operation, to the region of the umbilicus by suture, to secure a low drainage point, taking advantage of the fact that adhesion after operation was the rule, to secure fixation at a more favourable situation. These three cases have continued in good health, but there are valid objections to the plan. In the seven cases which came to secondary operation, the adhesions were most marked. Gastro-jejunostomy in each case resulted in cures. In the remaining fourteen cases, cure resulted. In four cases, an ulcer was excised at the same time, with favourable result. Pyloroplasty has a small field of usefulness, but in performing it the later plan of Mikulicz should be adopted. The incision should be curved downward upon both the stomach and duodenum, much like the Finney method, the result being to give an increased calibre over pyloroplasty as ordinarily performed, and establishing better drainage lines.

"The principle in plastic union, established by pyloroplasty, is one of the first importance and widely used in surgery. It is especially valuable in choosing the line of closure after excising gastric ulcers, etc."

Another operation which may be performed in conditions of this kind, is the gastro-duodenostomy of Finney. Mayo* followed the plan of Finney in 58 operations, with 4 deaths and with 2 secondary operations. In his first-mentioned article he reports his first 46 cases with but 1 death, but in the next 12 cases there were 3 deaths. Mayo counts all deaths following in the hospital after operation, and so it does not seem quite fair to count two of the deaths, as one was from pneumonia after complete recovery and one from embolus due to an old endocarditis. Mayo states that subjects for this operation should be carefully selected; extensive disease, adhesions, a short gastrohepatic omentum, and especially the presence of scar-tissue, should be considered a contra-indication, as it is just in these varieties that gastro-jejunostomy gives the most satisfactory results. Two of the four deaths were due to suture leakage on account of tension in scar tissue.]

4. *Gastro-enterostomy.*—The object of gastro-enterostomy is to obtain a new channel for the discharge of the chyme, by performing an anastomosis between the stomach and the small intestines. As experience has taught us, this procedure overcomes, as a rule, the mechanical disturbance, whatever its cause may be. Relying upon a previous suggestion of Nicoladoni, Wölfler,[130] in 1881, performed the first gastro-enterostomy; Wölfler's operation consisted in selecting a section of the jejunum lying about 40 centimetres from the pylorus and drawing it over the transverse colon and great omentum; next the stomach and the jejunum are incised and the two wounds united by suture. Both Courvoisier and v. Hacker deviated

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* William J. Mayo, *l.c.*

[130] Wölfler, Centralblatt für Chirurgie, 1881, No. 45.

from this technique. The former[131] proposed making an incision in the gastrocolic ligament and the mesocolon, through which opening a high coil of the small intestine is drawn and brought into communication with the stomach by inserting it into the posterior wall of that organ. v. Hacker's[132] operation consisted in pushing the stomach and the transverse colon upward and severing the mesocolon at a point free of blood-supply. The edges of this incision are then fixed to the posterior wall of the stomach by means of a suture, after which a high loop of the small intestines is joined to the stomach at this site. Aside from the above methods, others have been suggested and brought into practice, to mention and explain which would lead us into too lengthy a discussion. Observations as to the advantages and disadvantages of the single methods are for the present still divided; the material is also still too small to reach a definite decision as to the variously mentioned modifications. With reference to the results of the operation, we may consider them comparatively favourable in the benign pyloric stenoses, and it may be anticipated that with betterment of technique, and perhaps also by obtaining the patient's consent for an early operation, the chances will be even better. The statistics naturally vary considerably. A comparison of various large groups of results (Chlumski[133]) shows that the mortality rate for gastro-enterostomy in benign stenosis is approximately 20 per cent. This percentage is decidedly increased in malignant stenoses.

Gastro-enterostomy possesses the widest range of usefulness of all the operations to be considered in the cure of ectasia. It may be employed under all circumstances, and in every form and genesis of that condition; for example, it may also be resorted to in atonic dilatation, in which, as far as I know, gastro-enterostomy has, however, only been performed twice (Jeannel,[134] v. Kleef[135]).

[Mayo's statistics of gastro-jejunostomy shows 421 cases. Of these there were 307 cases of benign nature, with 19 deaths (6 per cent.); of the malignant 114 cases, there were 21 deaths (19 per cent.).

E. G. Roos, Scranton, reports excellent results (unpublished) in a number of operations on the stomach; he usually prefers the anterior gastro-enterostomy, and the Kocher method, especially the so-called Y-method.]

5. *Resection of the Pylorus which has been Constricted by an Ulcer.*—This operation was first performed for an obstructing ulcer by Rydygier,[136]

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[131] Courvoisier, Centralblatt für Chirurgie, 1883, No. 49.

[132] v. Hacker, Verhandlungen der deutschen Gesellschaft für Chirurgie, 1885.

[133] Chlumski, Beiträge zur klinischen Chirurgie, 1898, Bd. 20, S. 231.

[134] Jeannel, Archives provinc. de chirurgie, 1893, No. 1.

[135] v. Kleef, ref. in Berliner klinische Wochenschrift, 1893, No. 44, S. 1080.

[136] Rydygier, Volkmann's Sammlung klinischer Vorträge, 1882, No. 220, and Berliner klinische Wochenschrift, 1882, No. 3.

in 1881. The majority of surgeons condemn this procedure because of its accompanying dangers, uncertain functional results, and on account of the technical difficulties which it encounters. The operation is only justified in the comparatively few cases in which, during its performance, the character of the pyloric stenosis (benign or malignant) can not be established with certainty.

[William L. Rodman, referring to operations of this kind in the Journal of the American Medical Association of June 9, 1900, says: "If it were certain that individual operators had been as ready in reporting unsuccessful as they have successful cases, it would not be difficult to answer this question. It is, of course, the ideal operation and method of treatment; absolutely prevents malignant degeneration, and leaves the stomach in the best condition for future use. I have been able to collect from literature and extensive personal correspondence detailed reports of 40 pylorectomies, partial gastrectomies and excisions, with 6 deaths. Certainly this is a good showing when the nature of many of the cases is considered, operation in them having been undertaken for supposed malignant disease on account of a tumour with dense adhesions, and other conditions not conducive to a favourable operative result. This, furthermore, includes cases operated since 1881, when Rydygier performed the first operation of the kind. (This operation was soon followed (1882) by an equally successful case in the hands of Czerny.) Certainly the radical treatment of gastric ulcer has steadily gained in favour, and no one would now do as the editor of the journal in which Rydygier published his report of "The First Successful Excision of a Gastric Ulcer," namely, to write under it the sharp criticism, "and let us hope the last." Still, it cannot be said in candor that an operation with a mortality of 15 per cent. should be advised in all cases of gastric ulcer, but rather held in reserve for severe ones that will probably not yield to surgical measures less formidable in character."]

How does the stomach functionate after the operative treatment of a pyloric stenosis, whether the operation was a pyloroplasty or a gastroenterostomy? We have obtained satisfactory information on this subject from a large number of researches, including those of Dunin, Rosenheim, Mintz, Känsche, Siegel, v. Mikulicz and Kausch, Carle and Fantino, Heinsheimer, Kövesi, Débove and Soupault, Hayem, Mathieu, Mahant, Guedj, Terrier and Hartmann.[137] The results do not coincide in all respects, yet they deviate from each other only on unessential points. Following are the results obtained:—

1. The capacity of the stomach is, as a rule, diminished after gastro-

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[137] Quoted from Terrier and Hartmann, *l.c.*, S. 169.

enterostomy, especially in those cases in which the pyloric stenosis developed very rapidly (cauterisation, pyloric carcinoma).

2. The motility of the stomach is favourably influenced by pyloroplasty as well as by gastro-enterostomy, and returns more or less rapidly to normal; occasionally it falls below normal (Carle and Fantino), that is, hypomotility develops.

3. If hyperacidity exists, it gradually changes to normal or even below normal.

4. If motility and secretion have fallen below normal, they may again return to normal in the course of time.

5. Gastro-enterostomy is almost constantly followed by the presence of small amounts of bile in the stomach, which, however, causes no disturbance. After pyloroplasty this is a rarer occurrence.

The so-called vicious circle, that is, the permanent discharge of an abundant amount of bile into the stomach through the afferent limb of the jejunum, may be observed after performing the various methods of gastro-enterostomy. According to v. Mikulicz and Kausch,[138] the following ætiological factors are concerned: improper attachment of the intestinal loop as regards peristalsis; insufficient length of the afferent limb; formation of a spur; and finally, abnormal relaxation of the stomach and intestines. In rare cases rotation of the attached loop of intestines on its axis to 180°, or even 360°, may produce an obstruction to the passage. The usual result of these complications is continuous vomiting, which, if not soon controlled, leads to death of the individual. To avoid this undesirable condition, it was first proposed by Lauenstein[139] that the coil of jejunum which is to be sutured to the stomach be united with any convenient loop of the small intestines. Still better are the suggestions of Braun[140] and Jaboulay,[141] who established an entero-anastomosis between the afferent and efferent limbs of the loops of the jejunum, which is joined to the stomach, whereby the bile and pancreatic juice may flow through the more convenient channel, namely, the efferent branch of the jejunum, instead of into the stomach. This method is also employed with favourable results as a prophylactic measure by many surgeons, that is, they perform it simultaneously with the gastro-enterostomy. For other similar methods refer to text-books on special surgery.

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[138] v. Mikulicz and Kausch, *l.c.*, Separatabdruck, S. 29.

[139] Lauenstein, Fortschritte zu Ehren des 25 Jährigen Jubiläums von Prof. Ludw. Meyer, Hamburg, 1891.

[140] Braun, Archiv für klinische Chirurgie, 1893, Bd. 55, S. 361.

[141] Jaboulay, Archiv provinc. de chirurg., 1892, Bd. 1, No. 1.

Appendix.

Flow of Gastric Juice.

The Chronic and Continuous Secretion of Gastric Juice (Riegel); Gastrosuccorrhœa (Reichmann); [Gastrochylorrhœa, or Chylorrhœa].

Preliminary Remarks.—In 1882 Reichmann[142] discovered a symptom-complex which was characterised not only by peculiar subjective symptoms, but also by a continuous secretion of gastric juice, so that even in the fasting stomach digestive juice was secreted. Riegel[143] and his disciples, Honigmann[144] and Sticker,[145] also Jaworski,[146] van den Velden,[147] Bouveret and Devic,[148] Johnson and Behm,[149] and others, have done much to clear this subject. Nevertheless, it cannot be denied that in regard to frequency of the affection, its connection with other diseases, as well as the anatomical substratum, differences of opinion exist, the levelling of which can only be expected from future researches.

According to Reichmann,* the fully developed affection is rare; while others, particularly Riegel, Jaworski, Johnson, and Behm, are disposed to consider it as an exceedingly frequent occurrence. I believe this controversy can be explained by the fact that the said authors attribute too much importance to the presence of gastric juice in the fasting stomach. As soon as this symptom is emphasised too strongly, the frequency of the affection, as I will admit, jumps to an enormous height; but if the symptom-complex accurately described by Reichmann is regarded as a whole, even in the presence of a large number of patients, months may pass before a well-pronounced case of gastrochylorrhœa will be observed. Reichmann[150] observed only six typical cases in a period of several years.*

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* Reichmann himself doubts the frequency stated by Jaworski and Gluzinski (among 222 patients, 48 cases of gastrochylorrhœa), emphasising that the said authors did not always convince themselves of the presence of gastric juice in the fasting stomach.

[142] Reichmann, Berliner klinische Wochenschrift, 1882, No. 40; 1884, No. 48; 1887, No. 12, etc.

[143] Riegel, Zeitschrift für klinische Medicin, Bd. 11 und 12; Münchener medicinische Wochenschrift, 1884, No. 45-46; Deutsche medicinische Wochenschrift, 1887, No. 29; 1892, No. 21; Volkmann's Sammlung klinischer Vorträge, 289, 1886; Deutsche medicinische Wochenschrift, 1893, No. 30-31; Die Erkrankungen des Magens, Wien, 1897, S. 341, etc. (There exhaustive bibliography.)

[144] Honigmann, Münchener medicinische Wochenschrift, 1887, No. 48, etc.

[145] Sticker, Münchener medicinische Wochenschrift, 1886, No. 32-33.

[146] Jaworski, Zeitschrift für klinische Medicin, Bd. 11, Heft 2 und 3; Münchener medicinische Wochenschrift, 1887, No. 7 und 8; Wiener medicinische Presse, 1886, No. 52; Wiener medicinische Wochenschrift, 1887, No. 49, etc.

[147] van den Velden, Volkmann's Sammlung klinischer Vorträge.

[148] Bouveret and Devic, La dyspepsie par hypersecretion gastrique (Maladie de Reichmann), Paris, 1892.

[149] E. E. Johnson and K. Behm, Zeitschrift für klinische Medicin, Bd. 22, S. 478, etc.

The differences of opinion as to the frequency of the affection may also, without doubt, be due to the fact that some accept gastrochylorrhœa as a disease, others as a functional disturbance. According to my opinion, which in the meantime also Rosenheim and particularly Hayem confirmed, gastrochylorrhœa is certainly in many cases a symptom, or perhaps more correctly a complication, of well-known organic gastric diseases, such as gastric ulcer, myasthenia, and pyloric stenosis. It ought to be stated, however, that gastric ulcer as such does not occasion gastrochylorrhœa, at least as far as my own experience goes, but only then when motor disturbances have been added.

It may be left undecided which is the prime cause, the supersecretion or the motor disturbance. The reported surgical observations (Rosenheim, Boas, Hayem, Terrier and Hartmann) speak in favour of the latter, but at the present time there can be no doubt about the close connection between the perverse secretion and the mechanic insufficiency, if observed without bias.

On the other side, it cannot be refuted that in some rare cases periodic flow of gastric juice may be observed as a primary anomaly of secretion. These conditions differ so much, however, from the fundamental points of those recorded that it will be not advisable to mix them together. We will therefore discuss them in the Chapter on Gastric Neuroses, and restrain ourselves in the following only to the treatment of the chronic flow of gastric juice.

As to the pathologic-anatomic stratum, we have to deal, according to the latest investigations of Korczynski and Jaworski,[151] Hayem,[152] Cohnheim,[153] Hemmeter,[154] Strauss and Meyer,[155] Albu and Koch,[156] with a special type of gastritis (catarrhus acidus, Jaworski; gastrite hyperpeptique, Hayem) associated with degeneration and destruction of chief cells, while the border cells remain intact. Interstitial changes have been observed also, besides the parenchymatous ones. This opinion appears to me well founded, as the occurrence of gastritis in cases of gastric ulcer as well as of pyloric stenosis represents a pathologic-anatomic experience known for a long time (Cruveilhier, Rokitansky, Bamberger, Klebs, Lebert, Orth, and others).

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* Among the typical cases of Reichmann I cannot acknowledge case IV as supersecretion: it was evidently a carcinoma of the duodenum, probably of the pars descendens duodeni.

[150] Reichmann, Berliner klinische Wochenschrift, 1887, No. 14.

[151] v. Korczynski and Jaworski, Deutsche Archiv für klinische Medicin, Bd. 47, S. 578.

[152] Hayem, Gaz. hebdom., 1892, No. 33-34; Allgem. Wien. med. Zeitung, 1894, No. 2ff.

[153] Cohnheim, Archiv für Verdauungskrankheiten, 1896, Bd. 1, S. 274.

[154] Hemmeter, *ibid.*, 1898, Bd. 4, S. 23.

[155] Strauss, Virchow's Archiv, 1898, Bd. 154, P. 529.

[156] Albu and Koch, *ibid.*, 1899, Bd. 157, P. 1.

Diagnosis.

(*a*) *Subjective Symptoms.*

The symptoms are pyrosis, eructation, occasional vomiting of copious and intensely sour masses, paroxysms of pain several hours after meals or during night time. Characteristic also is increased thirst and temporarily-occurring bulimia; in other cases appetite may be decreased. Bowels are, as a rule, more or less constipated. The tongue shows a varying picture; the pulse, as in many gastric affections, is rather slow; skin is dry. As a whole, the said symptoms are occasionally so markedly pronounced that a probable diagnosis may be based on them alone. But only objective examination yields real certainty.

(*b*) *Objective Symptoms.*

To them belong the proof of a mechanic gastric insufficiency, increased acidity after ingestion of food, and the bad digestion of starch; furthermore, the presence of more or less large quantities of a colourless or greenish gastric juice in the fasting stomach. The urine is also characteristic to a certain extent.

The stomach is, in the majority of cases, mechanically insufficient and accordingly frequently dilated, but in some cases occurrence of normal size has been reported. Sometimes there is an insufficiency of the first degree, other times that of the second degree. In the latter cases a benign pyloric stenosis is usually found. Or there is first a statis insufficiency, but under appropriate treatment stagnation ceases, and instead of it we find an accumulation of gastric juice. The following case belongs to this class:—

Otto G., 40 years old, harness-maker, suffering from stomach trouble for twenty-five years; the affection developed gradually and got worse in the last years. Patient complains of pressure and fulness after meals, after a few hours there occurs vomiting of some water and mucus, rarely mixed with food; the suffering is worse after the ingestion of fluids. Never vomiting from the fasting stomach, nor nausea, never real gastralgia, appetite, sour eructations; bowels regular.

Physical Examination.—Organs of chest and abdomen without abnormalities. Stomach: after ingestion of a glass of water, marked splashing two fingers' breadth below the umbilicus, epigastrium not tender. Air inflation: vertical diameter 19 centimetres and horizontal diameter 28 centimetres.

January 12th. Test-breakfast: very acid, copious fluid. Acidity, 65.

January 13th. Fasting stomach: two waterglassfuls of contents. Three strata. A large quantity of yeast and sarcinæ, pronounced HCl reaction.

Treatment.—Diet, strychnia, daily lavage.

January 25th. Lavage every other day; only fluid with large amount of HCl; no food remnants.

January 27th. Fasting stomach, 80 cubic centimetres of gastric juice; acidity, 58.

January 30th. No contents.

February 3d. No contents. Discharged as cured.

The acidity of the gastric contents is usually very much increased. For titrating the acid, 70, 80, 100 per cent. of decinormal solution of sodium hydrate is needed, and sometimes more, whereby these values represent essentially the amount of hydrochloric acid, as organic acids, at least after test-breakfast, scarcely come under consideration.

The bad digestion of starch depends also upon this supersecretion of hydrochloric acid. The contrast between digestion of albumen and starch cannot be demonstrated in a better way than by removing the gastric contents several hours after a test-meal. While meat remnants are encountered either not at all or only in slight quantity, large quantities of unchanged starch are found. Very expedient is the demonstration of bad amylolysis by means of diluted iodine solution; while under normal conditions one hour after the test-breakfast the filtrate of the gastric contents turns wine-red or slightly violet colour by the addition of the iodine solution, on supersecretion of HCl it will show a saturated blue colouring. Under the microscope this reaction can also be readily demonstrated.

The presence of sarcinæ and yeast has been described by various authors (Reichmann, Riegel, Jaworski, and others) as a more or less frequent ingredient of the sediment. This cannot be astonishing if the close connection of gastrochylorrhœa with motor insufficiency is considered. It need, however, not be mentioned that as long as no stagnation exists, or existed shortly before, these frequent companions of the stasis insufficiency are not observed.

The most important criterion for gastrochylorrhœa is the presence of more or less acid gastric juice in the fasting stomach; but this is not a pathognomonic sign either. For, as Rosin,[157] Schreiber,[158] Martius,[159] and others have shown, more or less large quantities of digestive acid secretion can be recovered by aspiration from the fasting stomach in persons with absolutely healthy stomachs. (Martius found 40 cubic centimetres, Boas even 100 cubic centimetres.) When is the quantity pathologic; but less than 100 cubic centimetres may be pathologic if the other symptoms correspond to Reichmann-Riegel's clinical picture. It deserves to be emphasised that the presence of gastric juice in the fasting stomach, according to my observations, scarcely produces pains or any other complaints. The same belong rather exclusively to the digestive period and depend accordingly on superacidity.

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[157] Rosin, Deutsche medicinische Wochenschrift, 1888, No. 47.

[158] Schreiber, Deutsche medicinische Wochenschrift, 1893, No. 30.

[159] F. Martius, Deutsche medicinische Wochenschrift, 1894, No. 32.

Reichmann, and Riegel after him, in their publications, demanded, for the assurance of the diagnosis, that after washing out the stomach in the evening, the patient should not eat or drink anything until morning. The examination is made on an absolutely fasting organ. I consider this method in typical cases of gastrochylorrhœa as irrelevant, because I frequently convinced myself that the effect is the same, whether lavage was performed in the evening or not. Lavage ought necessarily to precede only in cases of stagnation of contents; but just in such cases I must join the reflections of Schreiber, whether under such conditions the flow of gastric juice does not represent an artificial product.

The *urine* is distinguished through a deficiency of chlorides; frequently, however, (A. Robin, G. Lyon[160]) an excessive amount of phosphates was observed. The reaction of the urine, especially after copious vomiting or after frequently repeated lavage, is alkaline.

The *bowels* are in almost all cases very much constipated, dry, hard, hazelnut-like; in some cases, however, there is temporary diarrhœa.

Differential Diagnosis.

In the differential diagnosis it is not so much the question whether gastrochylorrhœa is present—this fact can, as a rule, readily be elicited from above-said criteria—but rather whether we have to deal with a pathologic symptom *sui generis*, or if the gastrochylorrhœa is a secondary condition. Most frequently we have to think of mechanic insufficiency, perhaps associated with gastric ulcer. If mechanic insufficiency is present, it is to be investigated, whether it is one of first or second degree, which makes an essential difference as to the prognosis and treatment.

Riegel correctly differentiates between superacidity and continuous flow of gastric chyle; patients with continuous gastrochylorrhœa have, to be sure, always superacidity, while, on the other hand, in the presence of superacidity an abnormal secretion of gastric chyle is frequently absent.

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[160] G. Lyon, L'Analyse du suc gastrique, Paris, 1890.

Furthermore, Riegel states that in simple superacidity the digestive process is rapidly accomplished, while in gastrochylorrhœa the same is usually considerably retarded.

Treatment.

The object of the treatment is to remove the cause of the glandular irritation, and when this is not possible, to remove the disturbances arising from the superproduction of acid quantities. If there are symptoms of an unhealed ulcer, an ulcer-cure according to the well-known principles is to be recommended. Besides ulcer, occasionally a chronic overdistension of the stomach may lead to atony and further gastrochylorrhœa; but as already stated, the most frequent is either spastic or organic stenosis of the pylorus. Whatever the cause may be, in cases of this kind the chief task of the treatment consists in the removal of the gastric motor disturbances. This can be done, first, through avoidance of excessive quantities of fluid, then through the method of small but frequent meals. These should consist of mixed food, but not contain too large a quantity of meat (about 100 to 150 grammes *pro die*). Fats in quantities corresponding to normal conditions may be allowed; but spices, such as common table-salt, pepper, mustard, onion, nutmeg, etc., as well as acids, usually used for salads and mayonnaise, etc., are to be limited.

In cases of motor insufficiency of the second degree, the measures mentioned (p. 515) may be employed. Reichmann and Riegel, with whom the majority of authors agree, recommend lavage in all cases; Riegel prefers the evening, while Reichmann, as far as I can observe from his communications, selects the morning.

For the cases of gastrochylorrhœa which, according to my opinion, are to be designated as such, I consider gastric lavage necessary only when stagnation is present. Occasionally stagnation and gastrochylorrhœa may be alternating; in such cases a few washings-out may be employed; in all other cases I consider lavage superfluous. I cured many patients from gastrochylorrhœa merely by suitable diet, while to this time all kinds of medicines and various physical measures had been tried in vain. Sometimes patients may be told to express the contents from the fasting stomach, as I had them do successfully, in a few cases. Even this may frequently be superfluous. The following cases, taken at random from a large number, are examples for the small machinery necessary for removal of these disturbances:—

P. H., 31 years old, official, had pneumonia when 12 years old; never ill otherwise. Suffering from the stomach for the last four weeks. Vomiting first of the ingested food and finally green mucus, almost daily about twice, and usually one hour after meals. The vomited material tastes sour. About 15 minutes before vomiting patient has cutting pain around his umbilicus. The ingesta are expelled without retching, nausea. Appetite not good, bowels constipated, but spontaneous. Sleep only for one or two hours. No headache, some emaciation. Patient complained of anorexia for two or three months before onset of the affection.

Physical Examination.—Heart, lungs without abnormalities. Stomach: splashing several fingers' breadth below umbilicus almost to symphysis.

Test-breakfast: amount after 1 hour 20 minutes, 230 cubic centimetres, of acidity 97 (pronounced secretion of HCl).

After test-supper (20th of April) in the early morning: 290 cubic centimetres of water-clear contents, containing exceedingly large amount of free HCl (no food remnants). Acidity, 117.5 (intense HCl reaction).

April	21.	Fasting stomach:	45 cubic centimetres,	very acid.
"	22.	" "	153 " "	acid 102
"	23.	" "	35 " "	" 60
"	24.	" "	78 " "	" 78
"	25.	" "	68 " "	" 81
"	26.	" "	5 " "	" (Strongly acid)
"	27.	" "	50 " "	" 68
"	28.	" "	60 " "	" 52
"	29.	" "	30 " "	" 54
"	30.	" "	60 " "	" 50
May	1.	" "	35 " "	" 32
"	2.	" "	No contents.	
"	3 to 5.	" "	" "	
"	6.	" "	60 cubic centimetres,	slightly acid
"	8.	" "	40 " "	neutral
"	15.	" "	12 " "	slightly alkaline
"	19.	" "	Without free acid	
"	24.	" "	" " "	
June	4.	Patient discharged as cured.		

In a few cases (Rosenheim, Boas, Hayem, Terrier and Hartmann) there was indication for surgical treatment after which the symptoms of gastrochylorrhœa ceased.

An important aid is sometimes the systematic administration of alkali. On account of usually existing atony the acidulous waters, as well as solutions of Carlsbad salt, and the mineral waters used in cases of superacidity (Carlsbad, Vichy, Neuenahr, etc.), are to be avoided and to be replaced rather by alkalies in powder form (particularly magnesia usta, magnesium ammonii-phosphoricum, sodium bicarbonate or citrate, etc.). Jaworski[161] recommends for this purpose an aqua alkalina, a stronger and a milder one. The first contains in 1 litre of water charged with carbonic acid, sodium bicarbonate, 8.0, sodium salicylate, 2.5, borax, 2.0; the milder one contains sodium bicarbonate, 5.0, sodium salicylate, 2.0, borax, 1.0; a half to one full glass of these solutions to be taken after each meal. If constipation is present, Jaworski[162] lately employs effervescent magnesia water, and that also in a milder and a stronger solution. The milder one has the formula:—

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[161] Jaworski, Therapeutische Monatshefte, September, 1897.

℞ Magnesiæ carbonici	5.0
Magnesiæ salicylici	1.0
Solve in aqua acid. carbon. impreg.	1000.0

Sig.: One-fourth to one-half tumblerful 15 to 30 minutes after each meal.

The formula of the stronger solution:—

℞ Magnesiæ carbonici	10.0
Sodii chlorati	5.0
Soda water	1000.0

Sig.: One to one and one-half tumblerful in the morning on an empty stomach, or in the evening at bedtime.

Reichmann recommends nitrate of silver in large doses (0.03 to 0.09) in solution or in gelatine capsules.

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[162] Jaworski, *ibid.*, January, 1901.

CHAPTER XIX.

Displacement of the Stomach and its Neighbouring Organs.[1]

(Situs Viscerum Inversus; Vertical Position of the Stomach; Descensus Ventriculi; Gastroptosis, Enteroptosis [Glénard]).

Preliminary Remarks.—Displacement of some intestinal portions or other abdominal organs is a frequent occurrence. As early as in the celebrated work of Morgagni "De sedibus et causis morborum," excellent observations about the displacement of the stomach may be found. [illegible] Johann Friedrich Meckel, Ruysch, de Haen, and Cruveilhier have described displacements of the stomach, and the latter author[2] states "que ces déplacements s'observent surtout chez les femmes par suite de l'usage d'un corset trop serré!"

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[1] *Bibliography.*—Besides the text-books of Special Pathology and Therapy, and of Diseases of the Stomach, compare particularly:—

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Féréol, Bulletin de la [illegible]icale des hôpitaux, 5 [illegible]7; 12 Novembre, 1888.

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Dujardin-Beau[illegible] de l'hôpital [illegible] January [illegible]

Trastour, [illegible]

James [illegible]

C. A. E[illegible] pa[illegible]

A year later (1853) Virchow, calling attention to the displacements of the abdominal viscera, referred them to localised peritonic processes.

But only through Kussmaul's well-known treatise the frequent occurrence of abnormal positions was confirmed. Kussmaul already pointed to the various types and their demonstration.

We must not forget L. Landau's excellent monograph on the floating kidney. Nevertheless, these abnormalities became common property of the medical profession only through Glénard's numerous and able treatises on enteroptosis. Through the publications of Ewald, Kuttner, Curschmann, Herz, Einhorn, Kelling, Fleiner, Meltzing, Meinert, Langerhans, Rosengart, Stiller, Strauss, and others, further important building-stones were furnished to this new doctrine.

The views about the development of the displacement of the abdominal viscera differ essentially at the present time. Kussmaul already regarded a standstill during a fœtal stage of development as responsible for the vertical position of the stomach. Litten, Rollet, Ebstein, Leichtenstern, Ewald, Kuttner, and Drummond expressed themselves with lesser or greater certainty for a congenital displacement of the right kidney or the other intestine, while Meinert, on the other hand, sees the chief cause for the displacements in deformations of the thorax. The same, it is

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L. Kuttner, Berliner klinische Wochenschrift, 1890, No. 15-17; 3 Aufl., Bd. 19, S. 374, etc.; Eulenburg's Realencyclopädie, 3 Aufl., Bd. 14.

Meinert, Ueber Enteroptose, Sonderabdruck aus dem Jahresbericht der Gesellschaft für Natur- und Heilkunde zu Dresden, 1891-1892; Volkmann's Sammlung klinischer Vorträge, N. F., 1895, No. 115-116; Zur Ætiologie der Chlorose, Wiesbaden, 1894.

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Langerhans, Archiv für Verdauungskrankheiten, 1897, Bd. 3, S. 312.

Meltzing, *ibid.*, 1898, Bd. 4, S. 101.

Rosengart, Zeitschrift für physikalische und diatetische Therapie, 1898, Bd. 1, S. 215.

[2] Cruveilhier, Traité d' Anatomie pathologique générale, 1852, Tome IV, P. 855.

claimed, if their causes (pressure through clothing, injuries through occupation, rickets) have influenced several generations, become hereditary, and that in either sex. They represent then not congenital but acquired anomalies.

In recent times Rosengart[3] most pronouncedly spoke in favour of the fœtal formation. Supported by reported cases and his own observations on infants' bodies, he convinced himself that enteroptosis shows a great analogy to the position of the viscera during fœtal, or even post-fœtal life. The enteroptosis, therefore, must be referred on the one side to a persistence of the congenital position of the viscera, on the other side to the retrogression to the congenital position, influenced by external causes. Rosengart, in confirmation of Glénard, considers the downward displacement of the liver as the chief cause of the displacement of the viscera. All factors which press the liver downward, whether acting from the center upon the diaphragm, or from the outside upon the thorax, and all diseases and changes which allow the liver to sink downward in consequence of relaxed abdominal walls, will accordingly have to lead to enteroptosis. Kuttner, Langerhans, and Hemmeter join Rosengart in his view advocating the congenital development, while Landau, Kuster, and others refute it. Other authors, particularly Stiller (see below), find in the enteroptosis a special constitutional anomaly.

Glénard derivates enteroptosis from a prolapse of the transverse colon in the region of the flexura colico-hepatica; the originally weak ligamentum colico-hepaticum becomes more relaxed, the section between colon ascendens and transversum loses its hold, and the first change of position has been made. A relaxation of the other ligaments and mesenterium follows, and along with it a dropping downward of the viscera attached to the mesenteria, namely, the stomach, liver, kidney, spleen; in one word, there develops a general splanchnoptosis.

In consequence of the coloptosis there develops in the fixed section a kinking and an impediment to the propulsion of the intestinal contents; finally, an enterostenosis. The section of the transverse colon situated beyond the stenosis contracts itself, and the wall becomes thickened and is palpable as a solid string (corde colique transverse). In consequence of a further prolapse of the jejunum there develops a stenosis of the duodenum, with consecutive gastric dilatation.

Fromont,[4] in France, and Ewald,[5] Landau,[6] Kuttner,[7] in Germany, protested against the genetic view of these conditions. Ewald particularly pointed out that the corde colique transverse is nothing else than the pancreas easily palpable, as a rule, in the presence of lean abdominal walls; and likewise Fromont states expressively: "Les considérations sur lesquelles il (*i.e.* Glénard) s'appuie pour arriver a établir son entité morbide sont purement théoriques et ne repondent à aucun des faits constatés par nous sur le cadavre."

In late years E. Meinert,[8] supported by an extended number of observations, attempted to prove that the anatomical foundation of chlorosis must be looked for in the visceral prolapse, particularly in the gastroptosis. Meinert found gastroptosis in all cases of chlorosis; as the cause of the gastroptosis the corset has been accused by Meinert. It appears to me that Meinert does not differentiate closely enough the term of chlorosis from that of anæmia, though, to be sure, the term "chlorosis" possess a certain elasticity. I very frequently observed anæmia associated with gastroptosis, whereby the latter probably represented the inciting cause. The view of Meinert has been combatted by Meltzing,[9] Leo,[10] Brüggemann,[11] and most recently by Rostoski.[12] The latter found gastroptosis only in twenty-six per cent. of chlorotic patients. As Rostoski showed, the much-employed method of inflation with carbonic acid or air may easily lead to errors.

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[3] Rosengart, *l.c.*

[4] Fromont, Anatomie de la portion abdominale de l'intestin, Thèse de Lille, 1890.

[5] Ewald, Berliner klinische Wochenschrift, 1890, No. 12-13.

[6] Landau, Berliner klinische Wochenschrift, 1890, No. 18, S. 412.

[7] L. Kuttner, *l.c.*, Berliner klinische Wochenschrift, 1890, No. 15-17.

[8] Meinert, *l.c.*

It must be concluded from these discussions that a sufficient and generally satisfactory explanation of the cause of the displacement of the intestine does not exist at the present time. I agree with Strauss that a satisfactory answer cannot be given to a general question. Each case may have a special cause. Undoubtedly the fœtal theory goes much too far in its generalisation. It may offer a satisfactory explanation for those cases in which other ætiologic factors are missing; it becomes, however, superfluous in cases in which evident mechanic conditions, in the evident sense of the meaning, play a part.

According to Kelling,[13] the following conditions have to be considered:—

1. The stomach may be pressed downward. The most frequent cause in this regard is the lacing. The liver, deviating downward, presses the pylorus downward and to the left side. Besides through the corset, the lacing may be effected by fastening the skirts (petticoat and drawers), and in the male by wearing a tight belt. In rarer cases, gastroptosis may arise from the low position of the diaphragm or from tumours, echinococcus, enlarged liver, hydronephrosis.

2. The upper organs of the abdominal cavity, namely, liver and stomach, may be drawn downward by their weight. This occurs when the volume of the abdominal cavity has become too large for its contents; for the full stomach is heavier than the coils of the gut, and it moreover loses its partial support in the latter.

Other causes may also lead to enteroptosis, such as kyphosis and lordosis of the vertebral column, traumatism, adhesive processes, great tumours, ascites in the abdominal cavity, treatment for reduction of obesity, laparotomies, etc. Generally the word expressed by Gegenbaur is valid: *the position and form of the stomach are a product of adaptation of the dilating organ to the given conditions of space in the abdominal cavity.*

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[9] Meltzing, Wiener medicinische Presse, 1895, No. 50.

[10] Leo, Deutsche medicinische Wochenschrift, 1896, No. 12.

[11] Brüggemann, Inauguraldissertation, Bonn, 1895.

[12] Rostoski, Münchener medicinische Wochenschrift, 1900, No. 40.

[13] Kelling, *l.c.*

There occur quite a number of displacements of the stomach; only the following have a greater practical interest:—

1. *Situs viscerum inversus,* whereby the stomach is situated on the right side, and the liver on the left side; the cardia is turned toward the right, the pylorus toward the left. This exceedingly rare anomaly usually does not offer any particular difficulties for diagnosis, especially on application of gastric inflation by means of air or carbonic acid, of gastro-diaphany or Röntgen rays.

2. *Vertical Position of the Stomach.*—This condition occurs either congenitally or acquired, and is observed particularly in women. Tight lacing is ætiologically accused. In such cases the pylorus is situated lower and more to the left. As a rule, atony of the stomach is associated with the vertical position. Diagnostically, it is important that the horizontal diameter of the figure on percussion is smaller than normally; furthermore, the area of splash sound, if present, is cut off more or less in the median line; finally, on inflation with air or carbonic acid the protuberance is restricted exclusively to the left side of the body. In doubtful cases the diagnosis may be ascertained through transillumination or, as I observed, through tube palpation. Special practical importance is to be attributed to the vertical position of the stomach only in so far as it may easily give cause to motor disturbances.

[Eckwurzel[14] found, on post-mortem examination in a woman, the lowest part of the loop, corresponding to the pyloric valve, in the median line, midway between the umbilicus and the pubic symphysis. The stomach and duodenum were closely connected throughout the whole length of the loop by an expansion of the gastro-hepatic omentum and some areolar tissue. Duodenum was normal except for the length of its ascending portion, which, instead of being two inches, was eight inches. Her deformity was congenital, and never caused the slightest inconvenience during life.]

3. *Displacement of the Stomach Upward.*—This is usually of but inferior significance. Generally only the fundus is affected. Among the causes of a higher position of the stomach: pleuritis on the left side with retraction of the lungs. (Cases of this kind are rare; Fleiner reports one case in his book on Gastric Diseases.) Furthermore, influences limiting the space of the abdominal cavity (meteorism, ascites, abdominal

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[14] [George Macy Eckwurzel, Philadelphia Medical Journal, Feb. 3, 1900, P. 311.]

tumours, pregnancy, etc.) may push the stomach upwards. According to Fleiner, the stomach may be displaced upwards by the effects of lacing. He even mentions the occurrence of stomachs displaced upwards in men wearing belts (officers, laborers).

In cases of upward displacements of the stomach there may occur loops and kinks at the cardiac œsophageal portion whereby the ingestion of food is possible from above, while eructation and vomiting are made difficult or impossible (Fleiner) while the patient is in a recumbent position.

The diagnosis of upward dislocation of the stomach can be made only through a painstaking percussion of the costal fundus section while the patient is in a recumbent or erect position. In practice, however, too little attention has been paid to the occurrence. In an interesting case of upward displacement of the stomach with subsequent dilatation after an ulcer, S. Rosenstein[15] failed to make the diagnosis, diagnosing, instead of it, pneumothorax, because all symptoms of the latter were present. Fleiner[16] states that one must not conclude, from an upward position of the fundus and a simultaneous dislocation of the heart to the right, that there is a dilatation of the stomach, but must trace the course of the greater curvature, *i.e.*, its distance from the upmost fundus border, and must try, if possible, to determine the location of the pyloric part.

4. *Displacement of the Stomach Toward the Side.*—This anomaly is to be considered exclusively as a symptom of displacement caused through tumours of the spleen (leukæmia, malaria, etc.), through high position and overfilling of the flexura coli sinistra. Localised displacement of the pylorus arises from adhesive processes or through enlargement or ptosis of the liver.

5. *Displacement of the Stomach Downward. Descensus Ventriculi.*—Gastroptosis (Glénard) is the most frequent form of gastric displacement. We will limit ourselves in the following exclusively to gastroptosis.

As the descensus ventriculi is frequently associated with low positions of other abdominal organs, we have to consider the same also.

Very rare is the *total* gastroptosis, as the attachment of the lesser curvature acts as a limitation. The most frequent conditions are *partial* ptoses.

Diagnosis of Gastroptosis.

Subjective symptoms: those of the intestinal tract and nervous system. Objective symptoms: the demonstration of the displacement of the organs and its course.

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[15] Rosenstein, Archiv für Verdauungskrankheiten, 1896, Bd. 2, S. 161.

[16] Fleiner, Lehrbuch d. Krankheiten d. Verdauungsorgane, 1896, Bd. 1, S. 206.

(a) *Subjective Symptoms.*

Above all, it deserves to be mentioned that gastroptosis, even at an advanced stage, may exist without any symptoms. The frequency of this occurrence must of course remain unknown to even the most expert observer; but the absence of clinical symptoms in visceral ptosis is undoubtedly by far more frequent than was formerly believed. Enteroptosis as *entite morbide* has been decidedly exaggerated by Glénard, and also by other writers. It is, therefore, high time to give expression to the fact that enteroptosis as such remains probably always latent, and produces symptoms only when complications are added, such as traumatism, immoderate exertions, errors in diet, constipation, abuse of corset, etc.

The digestive disturbances, as far as they pertain to the stomach, consist, in cases of enteroptosis, of pressure, fulness, changing appetite, and are characterised from other disturbances through their irregular, paroxysmal condition, dependent at times upon the digestive proceeding and the quality of the ingesta, and independent from them at other times. Besides pressure and cardialgia, there also occur other abnormal sensations in the epigastrium, such as that of heat or burning. Eructation, pyrosis, regurgitation of food, even vomiting are often reported, but they are not typical for all cases. The gastro-enteric disturbances are lessened through bodily and mental quiescence, rest in bed, avoidance of psychical emotions.

Very characteristic for the gastro-enteric displacement is the habitual constipation. This is the rule, though in rare cases diarrhœa may sometimes occur, alternating with constipation. The abdomen is usually more or less distended; patients complain of flatulency.

In a great number of cases (about 27 per cent.) I observed the occurrence of membranous shreds in the stool, which did not always pass with severe pain.

Hereby it could be stated that the transverse colon, as well as other sections of the colon, were considerably displaced. I believe that in such cases the colitis membranacea is caused by the abnormal kinks and flexures at the affected section of the colon, which finally lead to a real and more or less extended catarrh of the colon. The following is an example of this kind:—

Mrs. M. T., 38 years old, was always well until her first confinement, 17 years ago. Afterwards abdominal complaints, continuing all this time, with exception of small intervals of relief, in spite of appropriate treatment. Five months ago patient took ill; severe pain in the left side. She observed mucous shreds in the stool; constipation; no pain.

Physical examination shows the following conditions: stomach has descended, the greater curvature deep below the level of the umbilicus, the lesser curvature at the level of the umbilicus. The right kidney is somewhat displaced (first degree). On inflation of the colon, the position of the transverse colon entirely below the umbilicus; its outlines are seen ascending from right below to the left above, in shape of festoons. In the region of the sigmoid flexure, marked tenderness. Moreover, patient suffers from left-sided parametritis and endometritis. On examination, the evacuated membranes correspond as to form and clinical condition to those in cases of colitis membranacea.

Sometimes the habitual constipation leads to ordinary intestinal catarrh with unformed and mucous discharges or stools, which are covered or mixed with large quantities of glassy and ropy mucus, or conditions of colitis membranacea alternate with common mucous colitis.

The symptoms from the *nervous system* are partly of local, partly of general nature. The former consist of more or less severe pains, particularly on exertion, on bending, or rash movements, and are evidently occasioned by dragging of the nervous plexus connected with the displaced organs. An almost typical, seldom missing symptom, is pain in the small of the back, which, nearly always present to a slight extent, may, particularly under the influence of movements and difficult exertion, reach an exceedingly painful degree.

Among general nervous symptoms we mention headache, which is frequently complained of, change of complexion, increased pulsation of the blood-vessels, especially of the aorta and its chief branches (*epigastric pulsation*). The latter symptom especially is not only exceedingly unpleasant to the patients, but troubles them to a high degree as if they had a pulsating tumour or something similar.

Of greatest importance are *nutritive disturbances,* which are reported in almost all typical cases of gastroptosis. The patients have usually lost considerable weight and adipose tissue, show scaly skin, cold extremities, get easily tired on the slightest bodily exertion; in brief, exhibit to the layman and frequently enough also to the inexperienced physician the picture of a grave organic disease. In less pronounced cases the nutritive condition is not quite so unfavourable; nevertheless, the majority of the patients are in a state of chronic subnutrition.

The causes are varied: fear of ingestion of food (sitophobia) on account of suffering, self-prescribed and stubbornly exercised abstinence as a remedial measure, restraint as to diet, prescribed by the physician. These improper measures are followed by further consequences: the patients continue to lose flesh, lose the appetite altogether on account of the one-sided food, constipation is enhanced and contributes much to the general malaise. There develops a vicious circle, which, as mentioned, may reach the most perilous degrees of inanition, unless an energetic reversion is brought about by the earnest intervention of the physician who recognises the condition.

The diuresis may be disturbed or be irregular if one or both kidneys are displaced, as for instance, by temporary kink of the ureter oliguria, or even anuria may occur, to be followed later by corresponding polyuria. If chronic kinking exists, real hydronephrosis may result.

(*b*) *Objective Symptoms.*

The objective symptoms refer principally to the proof of the displacement of the affected organs. The demonstration of the dislocated kidney on bimanual palpation is not difficult. Four degrees can be fittingly distinguished. (Litten, Ewald, Kuttner.)

1. The kidney shows only respiratory movability without being displaced.

2. The kidney shows dislocation of the first degree, *i.e.*, half or two-thirds of the kidney is palpable; it usually shows respiratory movability, is movable by the hands, and is displaced more or less forward.

3. The kidney shows dislocation of the second degree, *i.e.*, is palpable in its entire extent, is easily movable, shows respiratory movability, and lies close to the anterior abdominal wall, or at least can be approached near to it.

4. The kidney is dislocated and fixed, *i.e.*, the organ is displaced and immovable.

In accord with James Israel, we do not consider the first-named form as pathologic, though an abnormal respiratory movability may lead to a pathologic ptosis.

The formerly used term "floating kidney" is usually avoidable, as the above-named designations characterise the state of affairs better and more fully.

In regard to the *liver* also, various degrees of dislocation can be differentiated. In the first degree a more or less large segment of the liver can be palpated below the costal arch, while the upper border of the liver also has come correspondingly downward. In the second degree the greater part of the liver is felt to be situated in the abdominal cavity, and its contour can be easily palpated; the dulness over the liver is either entirely absent above the costal arch, or shows but a narrow zone. In the third degree the liver is situated entirely in the abdominal cavity. According to Einhorn,[17] with whom I essentially agree, five groups can be differentiated as to symptoms: (1) symptomless cases, in which the floating liver does not occasion any molestations; (2) dyspeptic cases with vague complaints connected with a sensation of weakness and many nervous symptoms; (3) hepatalgic cases, in which pain is nearly always present in the right side of the abdomen; (4) cases in which liver colics occur in paroxysms quite similar to those in cholelithiasis; usually no icterus present, though it may occur in rare cases; (5) asthmatic cases, in which a feeling of fulness and oppression in the upper abdominal region connected with slight dyspnœa appears.

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[17] Einhorn, Zeitschrift für diätetische und physikalische Therapie, 1900, Bd. 4, Heft 2, P. 99.

As Einhorn correctly emphasises, cases of displacement of the liver (the term "floating liver" is just as inappropriate as that of "floating kidney") are not at all rare.

The displacement of the *spleen*, which represents the rarest form of splanchnoptosis, has, on an average, slight clinical interest; but occasionally it makes considerable trouble, in consequence of passive congestion and enlargement brought about through the displacement. Here, too, different degrees can be distinguished, analogously to those of the kidney and the liver.

On displacement of the *stomach* the lesser curvature becomes accessible to percussion and palpation (splash sound), the greater curvature sinks more or less below the umbilicus, the upper gastric border (gastropulmonary border) is not found, as normally, in the fourth intercostal space, but lower downward. In gastroptosis of high degree, coils of the small intestines are pushed above the lesser curvature, which may obliterate or efface the dulness of the left liver lobe.

On *inspection* alone we are frequently enabled to make the diagnosis of descensus ventriculi. The epigastrium, normally convexed and at the same level as the umbilical region, has sunk in more or less, while above the umbilicus a decidedly pronounced convexity exists. The outline of the greater curvature can readily be observed by the experienced eye, and frequently also the outline of the lesser curvature. Figure 59 shows a case of pronounced descensus ventriculi, with the distinctly visible contours of the stomach.

Where doubt exists between dilatation and ptosis of the stomach, the stomach is to be *inflated* either with carbonic acid or with air.

A method more appropriate for purposes of demonstration is the gastric *transillumination*, which in many cases of gastroptosis yields very convincing pictures (Fig. 60).

Loss of elasticity of the gastric walls is frequently coincident with the descensus ventriculi, principally because they are deprived of the protecting bolster of the transverse colon. *By no means, however, as I will state emphatically, is myasthenia present in all these cases. The motor function of the stomach, at least as far as it can be proved through our relatively* *coarse methods of examination, is frequently perfectly maintained, though a persistent splashing seems to indicate the retention of considerable remnants of chyme.* Insufficiency of the stomach is shown only in very advanced cases. I rarely observed a real stasis insufficiency in cases of enteroptosis with displaced kidney and liver. The former statement of Bartels and Litten, that gastric dilatations are frequently associated with displaced kidneys, depends on the fact of mistaking atony and ectasia. On the other hand, the stomach is frequently found in the state of ptosis in cases of stasis insufficiency, particularly in advanced stages of pyloric stenosis.

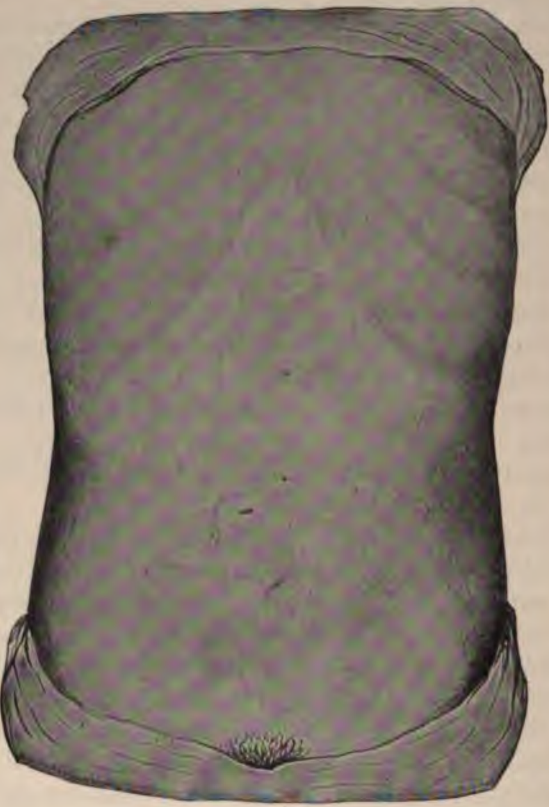

Fig. 59.

The examination of the stomach-contents by means of the tube may support the physical examination, in as far as it shows that the contents left the stomach on time; or if such is not the case, whether remnants are present to a large amount. The chemical examination proper of the gastric contents is of little value for the diagnosis of the gastroptosis; for there occur, for reasons not always readily discernible, all kinds of deviations from the normal condition of the gastric juice.

More important, perhaps, is the microscopical finding, as far as it shows the absence of fermentative germs (yeast, sarcinæ, bacilli of lactic and butyric acid, etc.). Sometimes the chemical examination may yield useful hints for the diet; we will revert to this point in the Chapter on Treatment.

Besides the displacement of the stomach, very often ptosis and kinking of some sections of the colon are found; the transverse colon and sigmoid flexure especially are involved. Displacement of the colon was demonstrated till now only through inflation of air by means of a tube introduced into the rectum, when the stomach and bowels were empty. Too large a distension of the colon, through which the outlines of the separate sections would be obliterated, has to be avoided. In normal cases an inflated area above or at the level of the umbilicus is to be observed; similarly, too, cylindrical swellings can be distinguished, corresponding to the colon ascendens and descendens, about one hand's breadth from the median line. In pathologic cases of displacement of the said sections of the

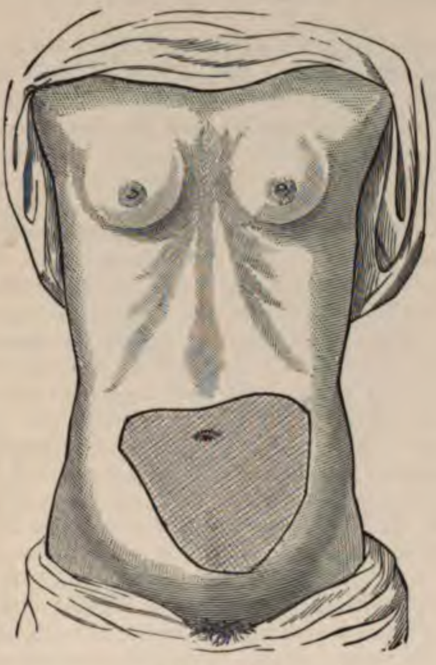

Fig. 60.

colon, the borders of the transverse colon move more or less downward, sometimes even down into the pelvis; those of the ascending and descending colon move considerably nearer to the median line than normally.

A second method of *determination of the position and size of the colon,* which I recently employed very advantageously, was described on page 90. It consists, briefly, of a gradual filling of the colon with measured quantities of water and eliciting the splash sounds arising in the corresponding sections of the colon.

Thus, for instance, I succeeded in demonstrating a colossal ectasia of the transverse colon in the following case:—

Bertha P., 49 years of age, seamstress, suffered for the last twenty years from obstinate constipation. All this time patient regularly used the most varied kinds of purgatives, and also enemata. The stool thus evacuated is hard as stone, covered by a glassy mucus. Occasionally there is an evacuation of solid membranous formations, accompanied frequently by severe pain. Besides, there is pronounced tympanites, pain in the small of the back, tenderness of the abdomen; appetite is good; occasional belching excepted, no gastric complaints. Patient underwent treatment for tapeworm five times, without result. It is noteworthy that patient, when 28 years of age, gave birth to a child, and there is now a retroflexio uteri. On physical examination, heart and lungs normal. The right kidney is *not* displaced. There is a ptosis of the stomach, the greater curvature a hand's breadth below the umbilicus, the lesser curvature at the level of the umbilicus. Within this area slight splashing. In order to elicit size and situation of the transverse colon, 300 cubic centimetres water are gradually introduced into the colon by means of a funnel, the stomach being empty. Marked splashing is demonstrated, and that in an area from two fingers' breadth above the navel to a hand's breadth above the symphysis. The largest vertical diameter of the splash-sound amounts to 13 centimetres. Pronounced succussion sound on changing the position. On inflation of the colon by means of air, a corresponding concavity in the said area is exhibited.

[In July, 1905, a case came under my observation which deserves mentioning. A man of very neuropathic character, who had been under treatment of quite a number of specialists in New York and Pennsylvania, and who had been under treatment for general neurasthenia, gastritis, and enteritis, came to my office with symptoms of very vague nature. He was very much addicted to cigarette-smoking, and not averse to drinks, especially as his work rather compelled him to entertain frequently. On examination I used the method recommended by Boas for determination of position of the colon, by gradually filling the colon with water, and the result is given in Fig. 61.—(*Col.* normal position, *Col.'* pathological position.)]

In connection with dilatation and displacement of the colon, frequently a state of contraction (Glénard made reference to it) of various sections of the intestine is met with, especially the cæcum, transverse colon, and sigmoid flexure. As already mentioned, Glénard designates this state of contraction as "corde colique." I suggest, instead of it, the more suitable name, "colospasm." We will not now discuss this state of contraction, which was observed also by myself in numerous cases; I will only limit myself to the statement that the diagnosis of colospasm may be easily made by means of inflation with air and also of introduction of water. The state of contraction then disappears instantly, returning after removal of the water or air. Colospasm also disappears after a copious evacuation of the bowels, but returns anew in the presence of coprostasis.

A true stigma in enteroptosis was pointed out a few years ago by Stiller.[18] This stigma consists of the so-called "costa fluctuans." Stiller could elicit in a large number of emaciated nervous dyspeptic persons with floating kidney, that their tenth rib is movable, *i.e.*, like the eleventh and twelfth ribs, not connected by cartilage with the costal arch, but entirely free and loosely attached to it by a ligament. Stiller even claims that the degree of enteroptosis, and still more certainly that of neurasthenia, may be concluded from the degree of movability of this rib.

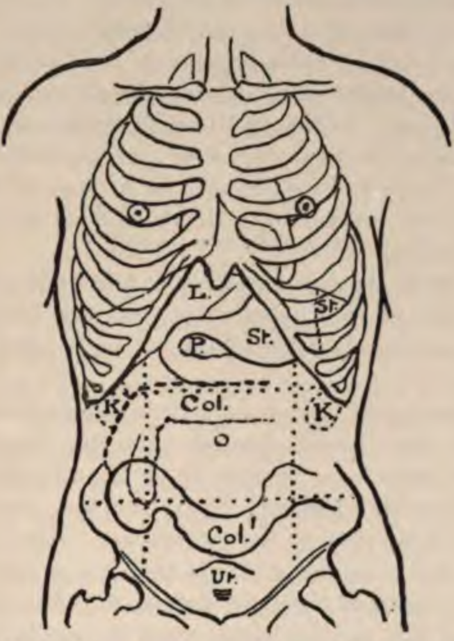

Fig. 61.—Coloptosis. (Observation of Dr. Bernheim.)

Various authors could confirm the observation of the floating tenth rib, and I also found Stiller's sign in many cases of enteroptosis. On exact investigations, made, on my instigation, by my former assistant, Dr. Walter Zweig, in reference to this subject, costa fluctuans was found, in

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[18] Stiller, Archiv für Verdauungskrankheiten, 1896, Bd. 2, P. 285.

100 cases not selected for this purpose, in 49 cases of nervous dyspepsia, in 8 cases of chronic glandular gastritis, in 9 cases of gastric ectasia (mostly pyloric stenosis), in 6 cases of chronic constipation, in 5 cases of gastric ulcer, and in 12 cases of other diseases of the most varied kind. As to percentage, the occurrence of costa fluctuans amounted to 65 per cent. in nervous dyspepsia, to 67 per cent. in enteroptosis; 34 per cent. of all cases with costa fluctuans were entirely free from all and any kind of nervous dyspeptic disturbances.

If further we consider that anatomic researches of Tandler[19] and Meinert[20] have shown that the tenth rib is more or less loose in a very great number of persons, even that it represents almost the normal fact, the value of Stiller's sign dwindles considerably. That it is felt extraordinarily easily and frequently in nervous dyspepsia and enteroptosis rests, according to my opinion, in the fact that in such cases we have usually to deal with emaciated persons, in whom it is especially easy to palpate the costal ends. The fact of the frequent occurrence of the floating tenth rib in neuropathic and enteroptotic individuals must be admitted, but not so its stigmatic significance.

[It is worth while to mention that in cases where the tenth rib is floating, the twelfth rib has usually become very small, sometimes so small that it has lost the character of rib and has become rather a process of the vertebra.]

Among other objective symptoms certain *points of tenderness* must be mentioned, which are present sometimes in the regions of the large abdominal ganglia, sometimes posteriorly on or near the vertebræ. The points of tenderness are characterised by the fact that they are deep-seated and usually correspond to the median line. They mostly are above the umbilicus, in rare cases below it. If the seat of painfulness is in the epigastrium, the thought of a gastric ulcer is forced upon our minds, but the multiplicity of the painful points speaks against it. On the other hand, it must not be forgotten that, as observed by me in several cases, also in enteroptosis with displaced kidney, an ulcer may manifest itself (see page 444). The pain points on the back, if present at all, are likewise not characteristic; sometimes they begin high up near the first or second dorsal vertebra and continue in a straight line down to the lumbar vertebræ; sometimes they begin lower down, and are interrupted by less sensitive or non-sensitive spots. In some cases pain points were observed only on the left side, in other cases on the right and left sides; and in the latter cases they usually did not correspond to one another.*

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* Compare Part I, P. 77 and 78.

[19] Tandler, Wiener klinische Wochenschrift, 1900, No. 8, P. 200.

[20] Meinert, Wiener med. Wochenschrift, 1900, No. 2.

(*c*) *The Course.*

The course of the descensus ventriculi is chronic, with frequent remissions and exacerbations. On account of the latter, already emphasised, gradual and excessive *nutritive disturbances* arise, so that a suspicion of a malignant disease, carcinosis or tuberculosis, may be excited. Signs of anæmia belong to the most frequent accompanying symptoms of gastroptosis. Great weakness, quick tiring, dislike for work, abnormal sensations are added to it. These symptoms are completed through changes in the nervous system, which likewise were already mentioned. Numerous exceptions from this typical course are frequent and cannot at all be explained by the objective examinations. Further investigation will have to give explanation. Characteristic and valuable to a certain extent, also, for the diagnosis, is the favourable influence of physical and mental rest, and *vice versa.*

Differential Diagnosis.

In the differential diagnosis are to be considered *pyloric stenosis, atony* (*myasthenia*), and *nervous dyspepsia.*

Above all, it deserves to be emphasised that the stomach in pyloric stenosis with consecutive dilatation is frequently found in a state of ptosis; dilatation of the stomach does not exclude gastroptosis. The question is only whether in a given case we have to deal with simple gastroptosis or a complication with stasis insufficiency. In the pronounced cases of grave insufficiency there is vomiting of sour and decomposed masses, which show the typical three layers; if vomiting occurs in gastroptosis, there is usually only a small quantity of undecomposed food. Correspondingly increased thirst and decreased diuresis are absent in cases of descensus ventriculi, in contrast to cases of ectasia. In the latter case, the state of nutrition sinks gradually; while in gastroptosis, suitable conduct of the patient being supposed, an improvement in weight and strength may be observed in a short time.

Still more important are the *objective symptoms.* We must determine through physical examination whether stiffening of the stomach or visible and palpable undulations are exhibited, which would speak for pyloric stenosis. If they are absent, there may nevertheless be stenosis, or also a grave gastric paresis without an obstacle on the pylorus. The surest decision whether the stomach is dilated is probably the diagnostic use of the tube on a fasting stomach after a previous test-supper. The stomach with insufficiency of high degree usually contains in the morning decomposed food remnants; the dropped stomach, whether complicated with myasthenia or not, in the morning is either entirely empty or contains, perhaps occasionally, more or less large quantities of gastric juice or bile, but by no means large quantities of food remnants.

It is of practical importance to know whether the stomach is only dropped or is also atonic, which, as before mentioned, is by no means the rule. In order to decide this, the examination of the motor function of the stomach, perhaps best by means of Leube's test-meal or Mathieu-Rémond's method, is the most suitable procedure.

We stated above (page 547) how enteroptosis in many cases may resemble the clinical picture of nervous dyspepsia; the differential diagnosis between the two affections may be difficult. As furthermore we have said already that gastroptosis, as well as ptosis of the kidney and liver, may occur without symptoms, we will not be able, if symptoms of a nervous dyspepsia are coincident with splanchnoptosis, to bring the former in correlation with the latter, though, of course, such a connection exists often enough. It is in practice, however, not of essential importance whether we have to deal with a primary or secondary or a complicating neurasthenia of the stomach. Still, it is possible, in an immoderate estimation of a perhaps slight gastroptosis, to overlook the presence of other causes of the nervous dyspepsia, as, for instance, entozoa or sexual affection.

As in a case of simple nervous dyspepsia, in the sense of v. Leube, the gastric motor function is usually well preserved, the examination according to the above-mentioned methods is of great importance. But the chemical examination of the gastric contents, in order to differentiate between the two affections, is of smaller importance, because in nervous dyspepsia, as well as in gastroptosis, the chemism varies considerably within wide limits.

Treatment.

By *prophylaxis* through regulation of the diet and of bowel evacuation (see below), also through appropriate bandages, we may be able to check an incipient splanchnoptosis, often still without symptoms, in its further progress. This is not a theoretically put-up postulate; for we are frequently enabled to elicit a beginning dislocation of the abdominal organs on complaints of vague abdominal disturbances. Where the same are pronounced, we must, above all, comply with the inciting indication. In some cases it can hardly be complied with; in other cases, however, it is possible. In habitual constipation, for instance, it must be attacked energetically, and the employment of strong exertion of abdominal pressure must be urgently forbidden. The corset must not be allowed, or at least is to be replaced by a suitable one.

The diet must first be nourishing and strengthening, and must exhibit *a rich amount of fat*. This requirement is best complied with by milk, and indeed quite satisfactory results may be seen from milk cures, if they are combined suitably with other nutritive substances. Milk cures, however, are suitable only in cases where milk in large quantities is well borne, and where no coarse motor disturbances in the gastric peristalsis are present. In the latter cases one administers, instead of milk, rather solid food rich in carbohydrates and fat. In regard to the former, Graham bread, because favourably influencing the intestinal peristalsis, is to be recommended; furthermore, almost all vegetables, excepting such as form much gas—I recommend spinach, cauliflower, carrots, chestnuts, white turnips, young green beans, young Brussels sprouts, asparagus, oyster plant, artichoke. Butter and cocoanut-oil are entirely sufficient as fat; plain butter sauces are not at all to be forbidden, as very often is still done. A very suitable food, distinguished by its high amount of fat, is v. Mering's [Hauswaldt] Vigor Chocolate, which has given me excellent service in diseases of this kind, as well as in others associated with more or less marked diminution of the nutritive state.

For the sake of influencing the evacuation of the bowels the so-called constipation diet showed itself satisfactory in most cases. Where this is not the case, oil enema or enema of emulsions of oil, codliver-oil, and soda solution have a sure effect in almost all cases.

Almost all sorts of meat may be allowed; wherever possible, the coarser ones are to be preferred. Eggs, as a rule, are not to be recommended, because, in my experience, they frequently produce flatulency.

Where the chemism according to the examination of the stomach has suffered decided disturbances, the diet must accordingly be regulated; but we must not overlook that if the motor gastric function is well preserved, the intestinal secretions may replace the deficient gastric secretions, so that an essential dietary restriction is hardly necessary.

The further therapeutic measures refer to the strengthening of the abdominal muscles and, if possible, also of the intestinal muscularis. This is reached by stimulating the function of the skin (bath, cold rubbing, douches), by abdominal massage, and by the extra- or intra-intestinal application of the faradic current.

The application of a suitable abdominal corset, as, for instance, Landau's or Bardenheuer's (see page 343), especially in persons with relaxed thin abdominal walls, is of undeniable palliative influence.

[In some cases I have used the method recommended by Achilles Rose with good palliative result. This method consists of the application of adhesive plaster fitted to the abdomen of the patient. The woman whose body is pictured in Figs. 62 and 63, was markedly relieved by this method.]

In rebellious cases of enteroptosis very favourable results may be had from a regularly instituted Weir Mitchell *rest-cure*, the details of which were discussed in the general part (page 310).

The *medicinal* treatment, compared with the mentioned remedial measures, steps more or less into the background. Glénard, leaning upon the doctrine of auto-intoxication of Bouchard, because in many intestinal displacements the absorption of many toxins is supposed to play a great part, recommended sulphur preparations, especially sulphate of magnesia and sodium and the mineral waters containing these salts. I hardly ever observed an advantage from these medicines, but rather harm. Indeed, in the course of time I omitted the pharmacologic treatment of enteroptosis entirely. Once in a while we may be compelled to prescribe a stomachic, a laxative, a sedative, or something similar; a persistent medicinal treatment, however, will certainly not satisfy the physician nor the patient.

[In recent years a number of operations for gastroptosis have been performed. The first operation of this kind was practised by Duret,[21] of Lille, in 1894; the second by Davis,[22] in 1897, and by Beyea, in 1898. After Beyea's first case, other surgeons, Bier,[23] Rosving,[24] Hartmann,[25] Coffey,[26] reported similar operations.

Beyea[27] briefly states: "The principle of the operation is that by placing three rows of interrupted silk sutures from above downward, and from right to left through the gastrohepatic and gastrophrenic ligaments, a single, broad, transverse fold or plication is formed in the ligaments, shortening these ligamentary supports and elevating the stomach to normal position. The normal ligaments are shortened and the stomach elevated to position without in the least disturbing the physiologic mobility of the organ." Beyea has operated upon several patients since his first operation—I was present at an operation a few months ago at the University of Pennsylvania Hospital; Edward Martin, of Philadelphia, and

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[21] Revue de Chirurgie, 1896, P. 430.

[22] Western Medical Review, October, 1897, P. 291.

[23] Deutsche Zeitschrift für Chirurgie, 1900, Bd. 56, P. 374.

[24] Archiv für klinische Chirurgie, 1899, Bd. 9, P. 812.

[25] Société de Chirurgie, April 19, 1899.

[26] Philadelphia Medical Journal, October 11, 1902.

[27] University of Pennsylvania Medical Bulletin, February, 1903; American Medicine, October 8, 1904.

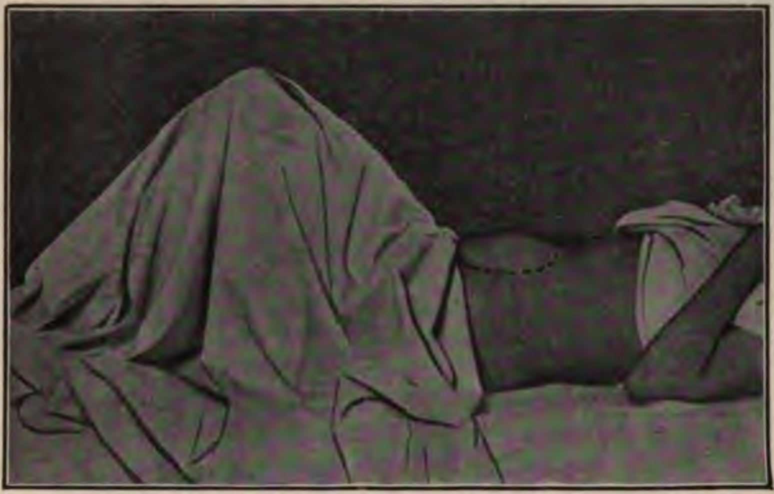

Fig. 62.—Gastroptosis (recumbent posture).

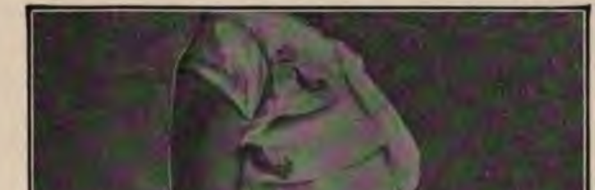

Fig. 63.—Gastroptosis (erect posture).

Charles Mayo, of Rochester, Minn., have practised Beyea's method in several cases.

According to Beyea's report, the surgical treatment for gastroptosis is without a doubt a remedial measure which ought not to be negected in suitable cases. While perhaps this operation is not absolutely indicated in patients who are able to afford all care and rest necessary, it becomes a necessity for many patients who are compelled to work for the necessities of life.]

CHAPTER XX.

Cancer of the Stomach.

(Carcinoma Ventriculi.)*

Preliminary Remarks.—Although carcinomata in general, on account of their fatal ending, excite our greatest interest, such is especially the case and to a high degree in carcinoma of the alimentary canal, and particularly so in that of the stomach. For, according to statistics of G. Heiman,[1] which refer to the total cancer morbidity of the Prussian hospitals in the years 1895 and 1896, the morbidity from gastric cancer amounts to one-third of all cancer affections in men; while in women, on account of the frequency of uterine cancers, the rate is considerably diminished. According to Reiche's[2] recent statistics, referring to cancer mortality of the city of Hamburg during 1872 and 1895, carcinomata of the stomach represent 50.2 per cent. of all carcinomata, and cancers of the total alimentary apparatus amount to the immense figure of 75.4 to 85.5 per cent.; the latter figure includes cancers of the liver, which, as Reiche correctly emphasises, originate in the stomach in the majority of cases. Even in women the proportion of gastric cancers amounts to 29.1 per cent.; and as the percentage of uterine carcinomata, the most frequent cancer in women, amounts to only 29 per cent., gastric cancer is even in the front rank in women.

If we still add to this fact that, according to Reiche,[3] the rate of mortality from cancer is slowly increasing in comparison with that from phthisis (the proportion of the latter to carcinoma is now 19 to 1, compared with 4.8 to 1 in 1872), and if we further consider that carcinoma, according to almost all recent observations (Hammerschlag, Kuttner, Strauss, Rütimeyer, and others). begins gradually to slide from old age to the strongest middle life, we have sufficient cause to pay special attention to intestinal cancers.

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* We discuss in the following only gastric cancer in its proper sense, *i.e.*, as far as it refers to the fundus and pylorus. Carcinoma of the cardia is nothing else than a deep-seated cancer of the œsophagus; it belongs, consequently, to the Chapter on Œsophageal Affections.

[1] G. Heiman, Archiv für klinische Chirurgie, 1899, Bd. 57, Heft 4.

[2] Reiche, Deutsche medicinische Wochenschrift, 1900, No. 7 and 8.

[3] Reiche, Münchener medicinische Wochenschrift, 1900, No. 39.

There is still another reason which shows gastric ulcers in yet another light at the present time, namely, the question of operability.

As long as cancer of the stomach was considered as an absolutely incurable affection, the simple elicitation of a tumour was sufficient for diagnosis; indeed, even the diagnosis "abdominal cancer" without further topographic decision could be regarded as sufficient, as a cure was impossible. At the present time, when removal of the tumour is considered as attainable, or at least as an aspirable object, our attention must be directed to the making of a diagnosis at the earliest possible time and as accurately as possible. From this standpoint, a patient suffering from cancer in general, as well as his constitution, his state of strength, and complicating diseases must be the subject of thorough discussion. Likewise, the local examination of the stomach must be made with a carefulness and completeness which, before the surgical era would have been superfluous, even if possible.

To a complete diagnosis belong, according to the present state of science: (1) the elicitation of the presence of a malignant tumour; (2) ascertainment of seat, size, and movability of the tumour; (3) ascertainment of position and size of the stomach; (4) ascertainment of metastases and ascites; (5) ascertainment of possible adhesion with contiguous organs.

According to these principles, the diagnosis will be discussed in the following:—

Diagnosis.

(*a*) *Subjective Symptoms.*

These may be exceedingly well pronounced in many cases, and in connection with the previous history may, even without examination, make diagnosis possible—diagnosis in a moment; in other cases the symptom-complex is so veiled that only a subtile examination, and sometimes not even then, may make it possible to elicit the condition. In the following we will describe the typical symptoms and will discuss the deviations in a special chapter.

Among the objective symptoms the following rank in first order:—

1. *The sudden beginning of the affection* and the accompanying progressive deterioration of the state of nutrition and strength. Cancer of the stomach begins in the midst of best health, particularly gastric health. v. Leube[4] accordingly states correctly that there is full cause to think of cancer of the stomach when the gastric affection in question happens in a person who for fifty or sixty years had a good stomach, which was never

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[4] v. Leube, Spezielle Diagnose der inneren Krankheiten, Leipzig, 1889, P. 552.

coddled and could endure all kinds of food. The reverse of this sentence, however, is not valid. Among 110 cases of gastric cancer which I compiled recently, there were about twenty-five times statements about previous *persistent* dyspeptic disturbances.[5]

2. *The Condition of Appetite and Thirst.*—Appetite is frequently (according to Brinton, in 85 per cent.) diminished; there exists particularly an almost insuperable aversion to meat. In other cases appetite varies, but always inclines toward anorexia. Occurrence of bulimia, as, for instance, described by Hanot,[6] was never observed by me, but not so rarely entirely normal appetite. Thirst is increased, particularly in obstructing cancers on or around the pylorus; in cancers on the curvatures there is no increase.

3. *Eructation* is a very frequent symptom in cases of cancer, on account of abnormal gas formation. The gases are odourless, or, on account of decomposing processes of the neoplasma, are exceedingly disagreeable, stinking; frequently there is complaint of strongly acid eructation.

4. *Pressure and Pain.*—In the beginning of the affection, as in chronic gastritis, there is only pressure in connection with ingestion of food. Later pain may follow, sometimes dependent upon the meals, sometimes independent of them. The pain begins in the pit of the stomach, or to the right or left, and radiates occasionally to the small of the back. The pain is described as drawing, burning, stinging, seldom as cramp-like.

5. *Vomiting.*—Vomiting is a frequent, but by no means constant symptom. It is rarely missing during the entire duration of the disease in carcinoma of the pars pylorica; but in cancer of the lesser or greater curvature it may be absent during the entire course, as I frequently have observed. Furthermore, as may be readily imagined, it is more copious in pyloric carcinomata than in those on the gastric curvatures. The vomiting is preceded by pressure or pain, nausea and salivation, and is followed by transient alleviation. The vomit, the particular qualities of which will be discussed in the Section on Examination of Gastric Contents, is described as bitter and sour; in some cases it is fœtid. Occasionally the vomit has the well-known "coffee-ground appearance," through admixture of blood from the ulcerating tumour. Formerly greater importance was attributed to this symptom than should have been. It is valuable for the diagnosis "carcinoma" if it occurs together with the other classic symptoms, particularly if observed repeatedly. That one ought not to content oneself with the "coffee-ground appearance," but must demonstrate a

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[5] Boas, Verhandlungen des 18. Kongresses für innere Medicin, 1900, P. 388.

[6] Hanot, Archives générales de Médecine, 1893.

bleeding by means of the hæmin test or guaiac test (see pages 223 and 224) does not need any discussion.

6. *Bowel movements* vary very considerably, depending on the location, variety, and size of the carcinoma. The movements may be normal, constipated, diarrhœic, and alternately constipated and diarrhœic. According to Fr. Müller,[7] in 35 per cent. of all gastric carcinomata diarrhœic evacuation is observed, an exaggerated condition, according to my experience.

7. *Amount of urine* is, partly on account of deficient ingestion of food and partly on account of vomiting and retention, diminished and concentrated.

(*b*) *Objective Symptoms.*

Among these there are to be considered inspection, percussion, palpation, and examination of gastric contents.

Inspection must extend first to the whole body, and then to the abdomen in particular. The former, because of the waste of the adipose and muscular tissue and of the scaly condition of the skin, furnishes us certain hints for the supposition of a malignant affection. The local inspection possesses, of course, a considerably greater importance. As the abdominal walls have usually lost their adipose tissue and the abdomen has sunk in, it is sometimes easily possible, particularly in women with enteroptosis, to observe quickly a prominence belonging to the epigastrium. According to my experience, this is particularly easy in carcinomata of the *lesser curvature.* If in such cases the patient is told to inspire deeply, the physician, particularly when standing at the head of the recumbent patient, will observe a cylindrical or sausage-shaped body descending more or less downward from the costal arch, in order to return to its former place on expiration. It is very important that the stomach is empty, or has been emptied for the sake of examination; for if the stomach is full, the tumour, as I frequently observed, may, on account of forward torsion of the greater curvature, be displaced completely backward, and thus may escape inspection and also palpation. Frequently in such cases the stomach is dropped; when such is not the case, the tumour is palpable only on deepest inspiration, and disappears entirely under the costal arch on expiration. In Fig. 64, a case of gastric cancer is pictured, in which the diagnosis was instantly made by means of inspection.

In carcinomata of the pylorus and the greater curvature, the tumour may be seen, but only under especially favourable conditions.

In carcinomatous pyloric stenosis, as in other varieties of stenosis, particularly if the stomach is very full, peristaltic and in rare cases even antiperistaltic waves may be observed to move from the fundus to the right, or *vice versa.*

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[7] F. Müller, Verhandlungen des Vereins für innere Medicin, 1888; Deutsche medicinische Wochenschrift, 1888, No. 22.

Percussion, compared with the other physical methods of examination, has but secondary importance; it facilitates only the ascertainment that on a certain spot a solid resistance is present which produces a dull or dull-tympanitic sound.

Palpation yields the most certain results of all physical signs. If a tumour evidently belonging to the stomach is found in a patient over thirty years of age with incipient or advanced cachexia, diagnosis is usually assured; but the tumour cannot always be recognised as belonging to the stomach. If a tumour is not palpable, it does not matter whether it has not attained a size necessary for palpation or is not accessible to palpation, one of the most valuable signs for diagnosis is missing. In spite of this a diagnosis can be sometimes made with a probability little short of cer-

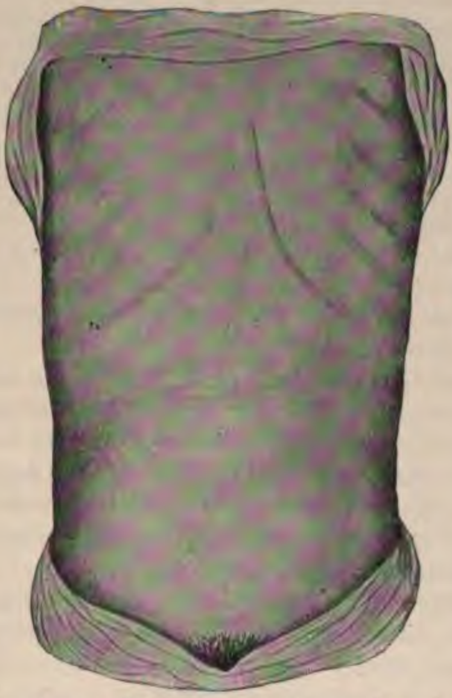

Fig. 64.—Gastric Cancer of the Lesser Curvature. (Observation of Boas.)

tainty, from the history, clinical course, and the examination of gastric contents. More about this further below.

For *seat* and *size* of the tumour there are of importance some statistic experiences which, in apparent agreement, show that the pyloric carcinomata are the most frequent, after them the carcinomata of the lesser curvature, and then after considerable interspace the carcinomata of the greater curvature, the diffuse infiltrates, and finally those on the anterior and posterior gastric walls.

Lebert[8] and Hahn exhibit the following statistics:—

	According to Lebert.	According to Hahn.
Pyloric region	51 per cent.	35.5 per cent.
Cardia	9 " "	23.5 " "
Lesser curvature	16 " "	15.9 " "
Greater curvature	7 " "	4.7 " "
Diffuse infiltrate	6 " "	12.3 " "
Posterior wall	4 " "	4.1 " "
Posterior and anterior walls	4 " "	?
Anterior wall	3 " "	4.1 " "

Hahn's figures, as readily seen, vary considerably from those of Lebert in several items.

The mentioned statistic figures are, however, in my opinion, to be considered with caution. They are based exclusively upon post-mortem examinations, whereby, of course, the original seat of the tumour cannot be elicited, or at least not with certainty. Very frequently it occurs that a carcinoma with an original seat on the lesser curvature emits its ramifications toward the pylorus. The pathologist as well as the surgeon, according to the general experience, will count a case in this stage among the pyloric cancers. The supposition that gastric carcinomata possess a special predilection for the orifices has been disputed by me for a long time, though without effect. Only within the last few years a revolution in this respect has begun to show itself with the pathologists and clinicians. Oscar Israel,[9] supported by repeated experiences, points to the fact that malignant tumours do not usually originate in the ostia, but end there. Similarly, v. Hansemann[10] states: "It must be emphasised that carcinomata of the pylorus itself are rather rare. The majority of the tumours begin above the pylorus and grow secondarily towards the same. This condition is perfectly plain if only one's attention has been turned to it."

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[8] Lebert, Die Krankheiten des Magens, Tübingen, 1870.

[9] O. Israel, Berliner klin. Wochenschrift, 1888, No. 31.

[10] v. Hansemann, Die mikroskopische Diagnose der bösartigen Geschwülste, Berlin, 1897, P. 179.

And finally, v. Mikulicz and Kausch,[11] in their excellent treatise on surgical gastric diseases, express themselves as follows: "The clinical observation shows that the primary pyloric carcinoma does not often occur, but, on the contrary, cancer develops primarily most frequently on the lesser curvature (perhaps 40 per cent.)." This agrees with my statistics based upon a material of 125 cases. I found the tumour 42 times on the lesser curvature, *i.e.*, 33.6 per cent.; on the pylorus, however, in 43 cases, *i.e.*, 34.5 per cent. To be sure, among the cases of cancer of the lesser curvature, as unavoidable, are also those cases in which either certainly or probably other parts of the fundus are affected by the process; but in all these cases the pylorus was certainly permeable, as shown by the course, or the operation, or the post-mortem. In spite of the above-mentioned important contradictions, the legend of the frequency of pyloric cancers migrates from one text-book into another, and, of course, does not gain anything in intrinsic truth, but the more in dogmatic strength.

Gastric tumours are situated either in the region of the pylorus, namely, to the right on the scrobiculus cordis, or in the same; or, if they are part of the lesser curvature under the left costal arch, palpable only on deepest inspiration, or in gastroptosis more or less deeply below the costal arch. In carcinomata of the greater curvature they are palpable at the level of the umbilicus in normal position of the stomach; in gastroptosis they are usually palpable below the same. Cancers of the pylorus, as well as those of the lesser curvature, may escape palpation, the former as soon as, according to the usual location of the pylorus, they are situated under the left liver lobe, the latter as soon as the lesser curvature, even on deepest inspiration, does not descend below the costal border. Carcinomata of the posterior wall are, of course, not accessible to palpation.

Of great importance is the *respiratory movability of the gastric tumours*, about which many erroneous views prevail. It is necessary to distinguish strictly between neoplasmata of the pylorus and those of the curvatures. The former, according to the anatomical situation of the pylorus, descends on inspiration not at all or but slightly; but if adhesive to the liver they move simultaneously with the normal liver excursion more or less downward.

Quite different are the carcinomata of the curvatures; as a rule, they show pronounced respiratory displacement. Moreover, they are frequently distinguished, as stated by Minkowski,[12] by *expiratory fixation*. If one grasps the tumour on deepest inspiration, it does not ascend on inspiration, but only when the grasp is released. Through this condition, on the one side these tumours can be easily differentiated from those of other organs, and on the other side the differentiation of cancers of the pylorus and the lesser curvature is facilitated.

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[11] v. Mikulicz and Kausch, Handbuch der praktischen Chirurgie von v. Bergmann, v. Bruns, v. Mikulicz, 1900; Separatabdruck, P. 132.

[12] Minkowski, Berliner klinische Wochenschrift, 1888, No. 31.

Rosenheim[13] called attention to the fact that expiratory fixation is observed, as a rule, in cases of respiratory-movable gastric tumours, but that the absence of expiratory fixation does not necessarily imply an adhesion with the liver. Such an adhesion, however, may be excluded if the tumour does not correspond, as to respiration, to the movement of the liver. As a case reported by Kundrat and Schlesinger[14] proves, this latter principle does not invariably hold good. In this case there was expiratory fixation in spite of extensive adhesion of the pyloric carcinoma with the posterior section of the lower liver surface. This fact appears very plausible, as the anterior parts remained unimpaired; the place of adhesion on the posterior part could yield the pivot for the abnormal movement.

The malignant tumours of the stomach are, as a rule, more or less tender to pressure: the algesimetric tenderness stands between that of gastric ulcer and that of glandular gastritis, and amounts to about two to four kilos. Besides tenderness anteriorly, occasionally a more or less severe, usually localised tenderness is also found posteriorly on the vertebral column, sometimes to the right, at other times to the left of it. As a rule, it is observed between the tenth thoracic and second lumbar vertebræ.

Besides the palpation, a very important diagnostic aid is *inflation of the stomach* by means of CO_2 or air (see page 105). The tumours of the pylorus and the lesser curvature act variously. The former, according to Minkowski, usually move to the right downward, or also (according to Rosenheim) occasionally to the right upward. Carcinomata of the lesser curvature, however, move gradually backward and disappear entirely on increasing inflation. The stomach undergoes a torsion, the greater curvature moving forward, the lesser curvature moving backward. The same torsion is sometimes exhibited under normal conditions of a full stomach; it happens that the tumour, as already mentioned, may entirely disappear and may become palpable only after emptying the stomach. I consider the latter symptom, which, according to my experience, occurs only in carcinomata of the lesser curvature, also as diagnostically important.

Besides better localisation of the tumours, inflation of the stomach facilitates an exact knowledge of the size and position of the stomach, which, in surgical operation, is of a certain importance for the direction of the incision. It must be mentioned that gastric dilatation is not, as might be assumed, the rule in cases of cancer of the pylorus. Stomachs of entirely normal size may be encountered. Still less frequently gastric dilatation is found in every case of stagnation, as one might expect. On the contrary, it is astonishing to find such proportionately small stomachs. Very much shrivelled and small stomachs are encountered, as may be briefly mentioned, and that not to the delight of the surgeon, in cases of cancer of the œsophagus and cardia.

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[13] Rosenheim, Deutsche medicinische Wochenschrift, 1894, No. 30.

[14] Kundrat and Schlesinger, Mittheillungen aus den Grenzgebieten der Medicin und Chirurgie, 1897, Bd. 2, Heft 5, P. 727.

Frequently in cases of cancer of the pylorus and lesser curvature, as mentioned above, a downward position of the stomach is observed; even vertical position of the stomach is not a very rare occurrence.

Inflation of the stomach may be advantageously supplemented under certain circumstances by means of "transillumination of the stomach." According to Kuttner and Jacobsohn,[15] in cases of non-palpable carcinoma of the wall, the transillumination, or rather the absence of transillumination, may become even diagnostically important. Yet the transillumination in these cases has not found general acknowledgment for the diagnosis of gastric cancers.

The decision whether a gastric tumour belongs to the pylorus or to the fundus can hardly be mistaken on careful physical examination. Besides this, there may be decisive factors in the exhibition of the "stomach stiffening" (page 74), or especially of visible and palpable gastric peristalsis, presence of grave stagnation, existence of gastric dilatation, oliguria, stubborn constipation, etc.

Besides the location of the tumour and the position of the stomach, the size and movability of the neoplasma are of importance. The smaller and more mobile the tumour, the better the chances for extirpation; this fact is especially valid in cases of cancer of the pylorus. It is of practical value to mention that, as Ewald has pointed out, cancers of the stomach appear larger to the palpating finger than they are *in facto*. But the reverse may occur also.

Is the tumour movable or adherent? The practically important adhesions are those with the liver, the gall-bladder, and the pancreas; they may complicate cases which by themselves are favourable for operation, so that surgical intervention must be entirely omitted or that only a palliative operation can be performed.

We refer in regard to adhesions with the liver to pages 567 and 568. For adhesions of a pylorus carcinoma with the pancreas, Angerer,[16] supported by observation of sixteen cases of this kind, stated the following symptoms as characteristic:—

If the carcinomatous pylorus is adherent to the pancreas, on inflation of the stomach it remains immovable or almost immovable; while if not adherent, the pylorus deviates in the regular way to the right (upward or downward).

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[15] Kuttner and Jacobsohn, Berliner klinische Wochenschrift, 1893, No. 39.

[16] Angerer, Archiv für klinische Chirurgie, Bd. 39, S. 378.

Are metastases present? This question can be answered only in some cases. The liver is implicated the most frequently, and as primary carcinoma of the liver is, as well known, exceedingly rare, tumours in the liver, occurring together with gastric tumours, are to be regarded as metastases. Metastatic cancers of the great and small omentum may be determinated under favourable circumstances by means of palpation, but in the majority of cases they are not diagnosed. In three cases I observed metastases on the umbilical aperture, in one case from a carcinoma originating in the depth of the small pelvis.

Cancers in an advanced stage frequently cause ascites and the accompanying physical signs, which, to be sure, are characteristic only when the accumulation of fluid has reached a certain amount.

The examination of the gastric contents is important in two respects: (1) on account of the motor disturbances occurring in cases of cancer of the stomach; (2) on account of the chemical disturbances. The former, according to my observations, are usually present in case of pyloric cancers, and in such a manner that the fasting stomach is rarely *entirely free* of food remnants. The same manifestation, however, occurs also, as shown by Boas,[17] Huber,[18] Martius,[19] Hammerschlag,[20] and others, in cases of cancers of the curvatures and fundus with such frequency that as a symptom it is diagnostically valuable. Since I paid special attention to this abnormality, I found among 125 cases of gastric cancers variously situated, 42 times, *i.e.*, in 33.6 per cent., considerable stagnation in the stomach.* This motor disturbance seems to me to depend by no means, or at least not solely, upon a dilatation of the stomach—inflation of the stomach with air or carbonic acid, as already emphasised, frequently showed entirely normal borders—but more upon a permigration of the cancerous tumour into the muscular layer.

The *chemical* disturbances are distinguished through the absence of free *hydrochloric acid* and through the presence of excessive *lactic acid.*[21]

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* If the frequent occurrence of motor disturbances is disputed by other authors, as, for instance, by Riegel, Kuttner, Schule, and others, these differences, like so many others in the study of gastric cancers, may be explained by the fact that many authors see their cases of cancer at an early stage, others at a medium stage, and still others at a very late stage.

[17] Boas, Zeitschrift für klinische Medicin, 1894, Bd. 25.

[18] Huber, Korrespondenzblatt für Schweizer Aerzte, 1896, No. 18.

[19] Martius, Zeitschrift z. 100 jährigen Stiftungsfeier d. med. chirurg. Friedrich-Wilhelms-Instituts, Berlin, 1895.

[20] Hammerschlag, Archiv für Verdauungskrankheiten, 1896, Bd. 2.

As to the much-discussed absence of hydrochloric acid discovered by van den Velden in 1879, it is generally acknowledged to be a frequent symptom. In my cases the sign was present in 77.5 per cent.; frequently free hydrochloric acid is found in the beginning, but disappears in the further course of the disease.

Unfortunately, however, the absence of free hydrochloric acid, as recent experience has shown, is just as little a characteristic sign for cancer of the stomach, as the reverse (hyperchlorhydria) is for gastric ulcer. According to my opinion we must differentiate a cancer of the pylorus or of the pyloric region from that of the fundus, regardless of the part at which the tumour has developed most. In cases of pyloric carcinoma the presence of free hydrochloric acid, or even hyperchlorhydria, is by no means a rare occurrence, even without a preceding ulcer. Among my cases I found free hydrochloric acid eighteen times in pyloric cancers, while in cancer of the fundus the same was found only four times. Evidently, this condition must be referred to the fact that pyloric cancers remain circumscribed for a long time and do not tend to pass the antrum pylori, while cancers of the fundus, extending without limits in all directions, destroy the glandular region proper in a short time.

Furthermore, as shown by Krukenberg[22] in 1888 and confirmed without any doubt by recent observations, the absence of hydrochloric acid by itself has been observed so frequently that there is no organic disease of the stomach—gastric ulcer not excluded—in which temporary or permanent absence of hydrochloric acid may not have been observed. It must, however, be conceded that the absence of free hydrochloric acid in patients who have reached the years disposed to cancer and show other signs of a malignant affection, perhaps exhibit even a resistance or a tumour, essentially supports the diagnosis.

Considerably more important is the *presence of an abnormal amount of lactic acid* in the gastric contents.

We must well distinguish between occurrence and formation of lactic acid in the gastric contents. The occurrence of lactic acid is not at all uncommon in gastric affections of the most various kinds, as we introduce lactic acid in greater or less quantities into the stomach by ingesting meat and fish, milk and its products, wheat and rye bread, cabbage, cucumbers, etc. *But new formation of lactic acid in the stomach occurs only under pathologic conditions.*

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[21] Boas, Deutsche medicinsche Wochenschrift, 1893, No. 17; Münchener medicinische Wochenschrift, 1893, No. 43; Berliner klinische Wochenschrift, 1895, No. 9; Zeitschrift für klinische Medicin, 1894, Bd. 25, Heft 3 and 4.

[22] Krukenberg, Diss.-Inaug., Heidelberg, 1888.

A test for lactic acid formation in the stomach, of course, can be made only if a *substance entirely free of lactic acid* has been introduced into the previously empty stomach. For this purpose I suggested a soup made of Knorr's oatmeal (without milk and butter). For purposes of daily practice, however, the test-breakfast* is satisfactory, as I have shown.

As a method for detecting lactic acid, Uffelmann's reagent may serve in first order (see page 188). It is true, this possesses, like all colour reagents, the Achilles heel of defective infallibility. The test, however, is entirely positive and practically satisfactory, if with a few drops of the filtrate an *intensely lemon-yellow colouring* of the very much diluted solution of liquor ferrichloride is shown. He who does not feel entirely sure is advised to make a control test by employing the same number of drops of a solution of lactic acid (1 or 2 per mille). If by the use of the gastric contents an equal colouring is obtained as by the application of the latter, there is no doubt of pathologic lactic acid fermentation. In some cases, though rare, of the solution of liquor ferrichloride (formation of iron phosphate), a cloudiness, depending upon a pronounced presence of phosphates, was observed, whereby abnormal lactic acid formation may be obscured. This is also the case in presence of *rhodanium* salts, which give a brownish-yellow colouring to the solution of liquor ferrichloride. As far as these admixtures occasion a doubt of existing pathologic lactic fermentation, it is well to employ the modifications of Kelling, Strauss, or Fleischer (see page 189); according to my experience, each one separately, or better all together, will positively solve the difficulty.

The aldehyde test suggested by me several years ago (page 192) has been invented and suggested as a quantitative laboratory method, and has been acknowledged almost generally as such. For practical purposes, however, this is too circumstantial, and usually unnecessary.

The formation of *lactates*, the chemically surest method, is also too complicated for clinical purposes.

The formation of lactic acid is conditioned undoubtedly through at least two factors, namely, stagnation and absence of hydrochloric acid. As to the latter, it needs to be mentioned that combined hydrochloric acid is sufficient to impede the formation of lactic acid. In such cases, as frequently observed by me, free HCl may be absent in the empty stomach, but under the influence of test-meals free hydrochloric acid, though in small amounts, may be found. Strauss[23] considers the ready adherence of lactic acid bacilli in the fissures and sinus of the tumour which scarcely participates in the peristalsis of the organ, as a decisive factor for the formation of lactic acid. While without doubt this fact is important, it is not decisive, as proved by the observations of lactic acid fermentation in cases of simple hypertrophic pyloric stenosis.

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* Whether Leube-Riegel's test-meal allows differentiation of the abnormally formed lactic acid from that inherent in the meat has not yet been investigated, as far as I know.

[23] Strauss, Zeitschrift für klinische Medicin, 1894, B. 26, S. 514.

According to Hammerschlag[24] an excessive diminution of enzymes, especially that of pepsine, plays a part favourable for lactic acid fermentation. Hammerschlag therefore states that the deficiency of pepsine is to be added as a third factor to those above named. Arth. Schiff, also, has observed the exhaustion of the enzyme production in cases of gastric cancer in contrast to benign gastric affection. I can likewise confirm the experiences of Hammerschlag and Schiff.

The pathognostic importance of the finding of lactic acid depends, on the one side, upon its frequency in cases of gastric cancer; on the other side, upon the occurrence and frequency of lactic acid formation in other gastric affections. As to the former, according to statistics of Arth. Schiff,[25] observations of Boas, Hammerschlag, Rosenheim, Lindner and Kuttner, Robin, Strauss, Kaufmann and Schlesinger, and Klemperer have shown in 261 cases of gastric cancer that lactic acid occurs in 73.5 per cent. of all cases. A higher rate was found by Croner,[26] namely, 78.5 per cent., and Rütimeyer,[27] 75 to 80 per cent. Still more pregnant is the importance of the finding of lactic acid if a larger number of gastric diseases of various kinds are compared with reference to it. According to Arth. Schiff, the statistics of Hammerschlag, Strauss, Schlesinger and Kaufmann, and Ekehorn show that *84.4 per cent. of gastric diseases complicated by lactic acid fermentation refer to gastric carcinoma.* Croner even found among 48 cases with positive finding of lactic acid 45 cases of cancer, *i.e.*, 93.75 per cent. Precisely the same result was obtained by Carle and Fantino in 78 cases of gastric carcinoma. As a counter-test I had in my polyclinic 100 cases of the various kinds of gastric affections with mere absence of hydrochloric acid compiled. There were only 31 cases of cancer of the stomach among them.

The importance of the presence of lactic acid tends unfortunately only toward the *positive test; absence of lactic acid does not exclude the presence of cancer of the stomach.* But even the constant presence of lactic acid fermentation does not, without exception, speak for a malignant tumour.

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[24] Hammerschlag, *l.c.*

[25] Arth. Schiff, Die Diagnose des Magencarcinoms, Sammelreferat nach den Arbeiten der Jahre 1894-1898; Centralblatt für die Grenzgebieten der Medicin und Chirurgie, 1898, Nos. 12 and 13.

[26] Croner, Mittheillungen aus den Grenzgebieten, 1899, Bd. 5, Heft 3, P. 405.

[27] Rütimeyer, Correspondenzblatt für Schweizer Aerzte, 1900, No. 21.

In the course of some years cases of abnormal lactic acid fermentation in nonmalignant gastric affections have been described by various authors (Ewald, Bial, Rosenheim, Strauss, A. Schmidt, v. Noorden, Lindner and Kuttner, Einhorn, Hammerschlag, Boas, and others). In the above-mentioned statistics of Arth. Schiff[28] observations of this kind have been compiled. There may be added a few others, as, for instance, those of Klemperer and Loewy, which refer to vitia cordis and oxalic acid poisoning, and therefore may be omitted in diagnostic or differential-diagnostic respect. The same is the case with an observation of Strauss (invagination of the small intestine with the colon) and that of Lindner-Kuttner, in which they had to deal with a perforative peritonitis after perityphlitis. In all these cases cancer of the stomach does not come into consideration at all. The majority of the remaining fourteen cases refer to chronic gastritis, atrophy with dilatation, and pyloric stenosis. Ewald emphasises particularly the frequent occurrence of lactic acid fermentation in cases of pernicious anæmia with gastro-enteric atrophy (eight cases). The differentiation of these cases from gastric cancer does not offer, as a rule, essential difficulties. The main difficulty and chief cause for deception are, in my opinion, the possibility of mistaking benign gastritis or atrophy complicated with primary dilatation or pyloric stenosis.

This possibility must certainly be admitted, especially for benign pyloric hypertrophy. But in such cases also, at least in a number of them, anamnesis and clinical course will prevent mistakes (see Differential Diagnosis).

While the finding of lactic acid does not possess an absolutely decisive importance, nevertheless the diagnosis obtains a high degree of probability. Indeed, various authors, supported by the constant finding of lactic acid, and, of course, simultaneously considering all the other symptoms, succeeded, to be sure not frequently, in a considerable number of cases (Boas, Rosenheim, Einhorn, Cohnheim, Huber, Hammerschlag, Alsberg, Ekehorn, Gockel, Soupault, Arth. Schiff) in making a diagnosis of gastric cancer without the presence of a palpable tumour. Whether such a diagnosis is also an early diagnosis will be discussed below.

The finding of hydrochloric acid, reported by a large number of authors in cases of gastric cancer even without ulcer origin, limits the diagnostic value of the lactic acid reaction to those cases in which the latter is positive. Nevertheless, there are still remaining sufficient cases in which, by means of lactic acid reaction, a diagnosis of carcinoma may be made, which *in the absence of a tumour was, as a rule, formerly impossible.* I mention the following two cases, with the finding at post-mortem examination:—

CASE I.—Albert R., iron-moulder, 40 years. Patient formerly healthy; took ill November, 1891, from dyspeptic disorders; all treatment without effect. April, 1892, he is referred to my policlinic for treatment, as a few symptoms indicate ulcer; use of stomach tube is omitted. After treatment with nitrate of silver, temporary improvement of ailment. Examination of gastric contents made then exhibits, after test-breakfast, against all expectation no free hydrochloric acid, but *marked presence of lactic acid; the same finding in the abundant contents of the fasting stomach in the morning.* From the stagnation and lactic acid reaction, and in respect to the course of the affection, the diagnosis of beginning cancer is made, though a tumour is not palpable, and the case is demonstrated as such in the post-graduate course (April, 1892). The same result is repeatedly found. Although a dilatation cannot be demonstrated anatomically, the patient is treated by means of lavage and like one affected with gastritis. Improvement of short duration. Patient is able to work. Two months later, August 26, 1892, patient feels again so sick that he returns to the policlinic. Tumour not palpable, stagnation and lactic acid reaction unchanged; patient is emaciated, vomited in the meantime repeatedly coffee-groundlike masses. He is treated with lavage for another month. Debilitation evident; he is referred to a hospital on September 29, 1892. Here he feels bad, vomits almost everything, and loses weight down to 80 pounds, while in the beginning his weight was 130 pounds. Diagnosis of cancer of the stomach accepted as certain. Beginning of December, 1892, he offers the picture of pronounced cancer cachexia, vomits all food; in the region of the gall-bladder a hard tumour, with slight respiratory movement, is palpable; it appears to be connected with the liver. On December 17, 1892, thirteen months after beginning of ailment, death. Autopsy shows an ulcerated pyloric carcinoma of the size of an orange, migrating to the lesser curvature and antrum pylori; almost entirely situated in the right abdominal part and covered to its larger extent by the liver. Metastases or adhesion with surrounding organs not present.

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[28] Arth. Schiff, *l.c.*

CASE II.—H., owner of restaurant at B., aged 46 years. Healthy till October, 1892. Since that time frequently complains of deficient appetite, very soon followed by vomiting. The vomiting very marked, so that no food could be retained. Rapid failing of strength. Loss of weight, 40 pounds in four weeks. Physical examination absolutely negative. At first neurosis suspected. End of October, 1892, clinical observation in my private clinic; result, no dilatation, no tumour, no painfulness. Quantity of urine during twenty-four hours is 300 to 400 cubic centimetres. No albumen, no sugar. Examination by means of stomach tube yields, in spite of the incessant vomiting, regularly remnants of unchymified masses to the amount of 50 to 100 cubic centimetres in the fasting stomach. Hydrochloric acid constantly absent, but intense lactic acid reaction (ether extract). Diagnosis of cancer of the stomach made. Under increasing debilitation death follows at his home on November 25th. Post-mortem, made by Dr. Vogt, chief surgeon of the army hospital at Bromberg, exhibits stenosing pyloric cancer.

Besides the palpable tumour, there is still another absolutely positive symptom, namely, the finding of *particles of the tumour* in the vomited material or in the gastric contents. Rosenbach[29] some time ago called attention to this sign, but it was neglected.

In many cases, the diagnosis, till then doubtful, may be ascertained by the finding of conglomerations of cancer cells in the lavage water. But as Reineboth[30] correctly states and Ewald already emphasised, errors and mistakes are not excluded. Only when a tumour is distinctly palpable, the diagnosis of a carcinomatous tumour may be made. The observations of particles of tumours have only recently been increased (Boas, Reineboth,[31] P. Cohnheim,[32] Hemmeter[33]). Reineboth found fragments in five cases of carcinoma; in two cases, on histologic examination, the diagnosis of cancer could be made with positiveness, once without palpable tumour; in three other cases the diagnosis remained negative in spite of the "fragments." Cohnheim compiled nine cases of gastric cancer with tumour particles from the cases in my policlinic. Just as in the cases of Reineboth, the diagnosis had been ascertained in our cases before the cancer particles were found. A decisive significance can be contributed to them only when other characteristic symptoms of gastric carcinoma are absent. Though in a few cases (as, for instance, that of Reineboth) the diagnosis may perhaps be made even without other clinical significant symptoms, the diagnostics of the stomach diseases will not be essentially influenced, because the finding of cancer particles, as Reineboth correctly emphasises, is evidently a late symptom. Hemmeter, for purposes of an early diagnosis of gastric cancer, suggested curettement of the gastric mucosa in order to find tumour particles or cell infiltration with karyokinetic figures. In forty-eight cases of gastric carcinoma he did not observe the least bad effects from this procedure. According to my opinion, quite apart from the fact that the method is not entirely harmless, the positive finding will be more or less a matter of chance.

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[29] Rosenbach, Deutsche medicinische Wochenschrift, 1882, No. 33.

[30] Reineboth, Deutsches Archiv für klinische Medicin, 1897, Bd. 58, S. 62.

A very important finding is, according to my observations, the occurrence of *blood* and *pus* in the gastric contents. Various kinds of hæmorrhages must be distinguished. Hæmatemesis, vomiting of pure blood or blood slightly mixed with food remnants, or decomposed blood (coffee-ground-like).

Similar results are found on examination of the gastric contents—either light-red blood, mixed with the contents or occurring on lavage of the stomach, or decomposed blood, whereby the colour may exhibit all tints, from a light-gray hue to the darkest blackish brown.

It may, however, occur also, as observed by me in numerous examinations recently, that the gastric contents apparently look quite normal, while by means of the guaiac test the presence of blood may be quite pronounced.

In the older text-books of Brinton and Lebert there are statements about the frequency of occurrence of blood in the stomach-contents. The former speaks of a rate of 42 per cent., mentioning that this is rather too low than too high, but Lebert finds graver bleedings only in 12 per cent. of the cases. In about 100 cases of cancer in which the gastric contents were investigated, I found 36 times statements about single or repeated hæmorrhages, but, according to my present experiences, these figures give but an imperfect impression of the frequency of particularly slighter hæmorrhages in gastric cancer. The significance of constant presence of blood is therefore, particularly in connection with the absence of hydrochloric acid and with lactic acid production, an exceedingly valuable sign, and we regard the statement of Carle and Fantino,[34] that the concurrence of these two symptoms can almost be considered as pathognostic for cancer, as approaching very near to truth.

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[31] Reineboth, *l.c.*

[32] P. Cohnheim, Archiv für Verdauungskrankheiten, 1896, Bd. 1, S. 274; "Festschrift Lazarus," April 1, 1899.

[33] Hemmeter, New York Medical Record, October 21, 1899.

The combination of blood and pus or the occurrence of pus alone is rarer (Boas, Cohnheim,[35] H. Strauss[36]). The former was found five times, the latter eight times among my cases.

It must be mentioned that there are cases of gastric carcinoma which even on most painstaking lavage exhibit constant presence of pus in the gastric contents. Apart from the microscopic examination, which, however, is only characteristic when pus corpuscles are found in dense conglomeration, it is the offensive smell of the stomach-contents which excites suspicion of presence of pus. P. Cohnheim calls attention to another variety, in which pus is embedded in mucus. According to Cohnheim, besides pus, red blood-corpuscles, glandular cells, tumour cells, and other textureless formations are found; the latter are considered by the above-named author as fragments of the tumour. The purulent mucus, in contrast to the pure pus, is not fœtid. As acute suppurations, occurring in cases of phlegmonous gastritis as a rule, may be excluded, and occurrence of pus in the gastric contents of benign processes is not observed, this symptom gains an invaluable significance in doubtful cases.

The pus undoubtedly is derived from necrobiotic decomposing cancer tissue, occurring particularly often in cases of readily exulcerating medullary cancers.

On *microscopic examination* of the stagnant gastric contents in cancer of the stomach, as a rule, numerous undigested muscle fibres are exhibited, sometimes with well-preserved transverse striation, as well as amylum and fat in larger or smaller quantities.

A striking picture of the carcinomatous gastric contents is formed through immovable *bacilli*, first observed by myself. They are long, filiform, frequently joining one another in angles; they appear in numberless specimens in the field of vision (Fig. 65). Particularly frequent, almost constant, they are found in stagnant, achlorhydric gastric contents containing lactic acid.

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[34] Carle and Fantino, Archiv für klinische Chirurgie, 1898, Bd. 56, S. 222.

[35] Cohnheim, *l.c.*

[36] Strauss, Berliner klinische Wochenschrift, 1899, No. 40.

These bacilli have been studied more accurately since then by Oppler,[37] Schlesinger and Kaufmann,[38] Strauss,[39] and Rud. Schmidt.[40] Schlesinger and Kaufmann were the first to succeed in establishing pure cultures of these bacilli upon flesh-peptone-agar with addition of carcinomatous gastric contents, then also upon beer-wort and glucose-agar. Strauss could also make a pure culture of the bacillus upon flesh-peptone-agar with carcinomatous gastric contents, with the addition of a small quantity of a sterilised solution of glucose.

The length of the bacillus is, according to Schlesinger and Kaufmann, 6 to 8 μ, but varies in the gastric contents as well as on the artificial culture media between

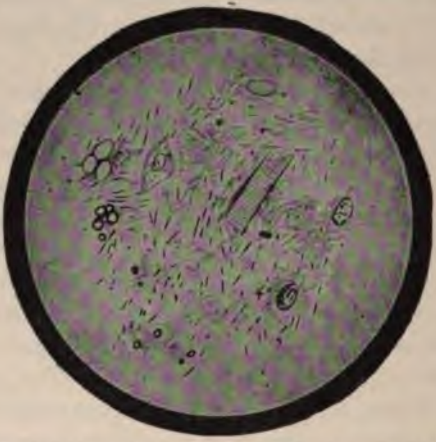

Fig. 65.—Long, Filiform Bacilli found in Carcinoma of the Stomach. (Observation of Boas.)

3 and 10 μ. Occasionally they grow as long, convolute, apparently disjointed filaments. Its thickness is 1 μ. The bacilli are, as mentioned, immovable, facultatively anaerobic, without formation of spores. On short staining with aqueous solution of methylene-blue, according to Strauss, some filaments, stained a light-blue, show granula of various size which accept the stain in a different manner, namely, intensely blue-red, and make a striking contrast to the remaining bacterial body. Strauss accepts these formations as plasmogenous corpuscles.

On staining according to Gram, no decolourisation follows through solution of potassium iodide.

The bacilli are capable of coagulating milk, but, above all, of generating lactic acid from various kinds of sugar. They may, therefore, quite correctly be designated as the inciting factor, or at least as one of the inciting factors, of the lactic acid formation in the stomach.

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[37] Oppler, Deutsche medicinische Wochenschrift, 1895, No. 5.

[38] Schlesinger and Rud. Kaufmann, Wiener klin. Wochenschrift, 1895, No. 15.

[39] Strauss, Zeitschrift für klin. Medicin, 1895, Bd. 28, S. 578.

[40] Rud. Schmidt, Wiener klin. Wochenschrift, 1901, No. 2.

The examination for lactic acid bacilli yields, in the original specimen as well as in the suspended drop, as a rule, a perfectly satisfactory picture. Rud. Schmidt[41] recommended Lugol solution as a suitable addition, whereby its being mistaken for the bacillus maximus buccalis (originating in the oral cavity), which is stained blue-violet through iodine, can be excluded. On the other side, amylaceous detritus is readily recognised by its blue colour resulting from iodine stain. As other similar bacilli (bacillus mesentericus) (R. Schmidt) show quite similar pictures, in case of doubt pure culture becomes indispensable.

The diagnostic importance of the lactic acid bacillus coincides essentially with that of demonstration of lactic acid. In this sense they form a mutual supplement, so that I always advise looking for the presence of bacilli besides the lactic acid. In doubtful lactic acid reaction, a positive finding of bacilli may perhaps be decisive. As Sternberg[42] recently found bacilli of the same appearance and with the same culture qualities in the stomach of a case of incarcerated hernia, there is nevertheless demand for caution in the valuation of the finding of bacilli.

Sarcinæ.—In the stagnant carcinomatous gastric contents occasionally sarcinæ, and frequently yeast are found. There is disagreement on the occurrence of sarcinæ in cases of gastric cancer; some writers consider the same as frequent, others as rare; eminent earlier investigators, as for instance Bamberger, attribute a diagnostic significance to the presence of sarcinæ. According to my investigations, sarcinæ are decidedly rare in gastric carcinoma with lactic acid fermentation. That lactic acid formation is unfavourable to the development of sarcinæ has undoubtedly been demonstrated in my laboratory by Oppler,[43] by the fact that pure cultures of sarcinæ which were poured into carcinomatous stomachs with lactic acid had disappeared at the end of twenty-four hours. Moreover, another fact is convincing: if in carcinomatous gastric contents, which in the beginning exhibit free or combined hydrochloric acid, the latter disappears, sarcinæ are usually replaced by lactic acid bacilli.

Symptoms of Second Importance.

1. *Anæmia and Cachexia.*

Carcinoma leads in a short time to cachexia and anæmia. Cancer cachexia has been spoken of, meaning a peculiar wax-yellow appearance accompanied with a high degree of emaciation. This symptom has but a limited diagnostic significance, occurring, as it does, in a pronounced manner only in late stages, at a time when diagnosis is no longer difficult. Moreover, cancer cachexia does not show any specific manifestations deviating from other diseases with severe debility, to mention only Addison's disease, cirrhosis of the liver, amyloid degeneration, certain cardiac diseases, etc.

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[41] Rud. Schmidt, *l.c.*

[42] Sternberg, Wiener klin. Wochenschrift, 1898, No. 31.

[43] Oppler, Münchener med. Wochenschrift, 1894, No. 29.

Anæmia is produced first through more or less extensive bleedings from the gastric or intestinal mucous membrane, second through disturbances of the alimentary assimilation, and finally through admission of toxins into the blood.

Anæmia may manifest itself through a moderate leucocytosis, which, however, is not characteristic of cancer; further, through diminution of hæmoglobin, which, according to Häberlin,[44] falls to about 50 per cent. of the normal amount. Similar statements have been made by Laker,[45] Leichtenstern,[46] Eichhorst,[47] and Osterpey.[48] Investigations of my own have likewise shown a considerable decrease of hæmoglobin in the majority of cases. As in other positive cases of carcinoma, the amount of hæmoglobin may give us a supplementary but by no means a positive criterion for cancer.

Parallel to the decrease of hæmoglobin there is frequently a decrease of the number of red blood-corpuscles (Laache,[49] Fr. Müller,[50] Oppenheimer,[51] Häberlin,[52] Osterpey,[53] and others). Yet the decrease of the red blood-cells cannot be accepted as specific, so that we likewise attribute but a certain diagnostic value to this anomaly.[54] According to F. Henry[55] it is claimed that the number of red corpuscles in cases of gastric cancer never sinks below 1,500,000 in the cubic centimetre, while in pernicious anæmia it almost always falls below a million.

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[44] Häberlin, Münchener med. Wochenschrift, 1888, No. 22.

[45] Laker, Wiener med. Wochenschrift, 1886, No. 18-20.

[46] Leichtenstern, Untersuschungen über den Hämoglobingehalt des Blutes in gesunden und kranken Zuständen, Leipzig, 1878.

[47] Eichhorst, Handbuch der speziellen Pathologie und Therapie, Bd. ii, 4 Aufl.

[48] Osterpey, Die Blutuntersuchung und deren Bedeutung bei Magenerkrankungen, Inaug.-Diss., Berlin, 1892; see also Berliner klinische Wochenschrift, 1892, No. 12 and 13.

[49] Laache, *l.c.*

[50] Fr. Müller, Verhandlungen des Vereins für innere Medicin, 1888, P. 378.

[51] Oppenheimer, Deutsche medicinische Wochenschrift, 1899, No. 42-44.

[52] Häberlin, *l.c.*

[53] Osterpey, *l.c.*

[54] Compare Part I, P. 264.

[55] F. Henry, Archiv für Verdauungskrankheiten, 1898, Bd. 4, P. 1.

Recently J. Schneyer[56] called attention to a supposedly important diagnostic sign, namely, the absence of *digestive leucocytosis* in cancer of the stomach, in contrast to benign pyloric stenosis and ulcer, in which latter cases leucocytosis is usually observed. According to the statement of Schneyer, this symptom is said to be independent of the location, duration of the cancer, and the chemism in the stomach. Schneyer's statement has been confirmed only by Hartung.[57]

On further researches of Hassmann,[58] Hofmann,[59] Sailer[60] and Taylor, Chadbourne,[61] Dolmatow,[62] Rencki,[63] Marchetti,[64] and others, it was shown, however, that on the one side, though much rarer, digestive leucocytosis is observed also in cases of gastric cancer, while on the other side in benign gastric affections (ulcer, gastritis, inacidity, neurosis) it may be missing. The state of digestive leucocytosis, therefore, is of no positive diagnostic importance.

Finally, the *specific gravity* of the blood is to be mentioned, which can be found in a safe manner according to the methods of Hammerschlag[65] or Schmaltz.[66] According to observations of Schmaltz and Siegel,[67] specific gravity of the blood is parallel to its amount of hæmoglobin. In carcinoma of the stomach, Peiper[68] found decrease of the density of the blood; I found the same in four cases. These findings also may enable us in a doubtful case to guide the diagnosis into the right track, but a decisive importance cannot be attributed to them.

2. *Enlargement of Supraclavicular Glands.*

In the General Part (page 86) we mentioned the swelling of the left supraclavicular glands and spoke of their slight diagnostic importance.

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[56] Schneyer, Internationale klinische Rundschau, 1894, No. 39.

[57] Hartung, Wiener klinische Wochenschrift, 1895, P. 697.

[58] Hassmann, *ibid.*, 1896, No. 17.

[59] Hofmann, Zeitschrift für klinische Medicin, 1897, Bd. 33, P. 460.

[60] Sailer and Taylor, ref. Archiv für Verdauungskrankheiten, 1898, Bd. 4, P. 88.

[61] Chadbourne, Berliner klinische Wochenschrift, 1898, No. 2.

[62] Dolmatow, ref. Archiv für Verdauungskrankheiten, 1900, Bd. 6, P. 96.

[63] Rencki, *ibid.*

[64] Marchetti, Settimana medica, 1898, No. 46; Ref. Centralblatt für innere Medicin, 1900, P. 401.

[65] Hammerschlag, Zeitschrift für klin. Medicin, 1892, Bd. 20, P. 444.

[66] Schmaltz, Deutsche med. Wochenschrift, 1891, P. 555.

[67] Siegel, Wiener klin. Wochenschrift, 1891, P. 606.

[68] Peiper, Das spezifische Gewicht des menschlichen Blutes, Centralblatt für klinische Medicin, 1891, No. 2.

ir views are held by Ewald,[69] Riegel,[70] Debet,[71] Troisier,[72] Soupault
abbé,[73] Lépine.[74] According to his renewed observations, Tarchetti[75]
his dissent to attributing a certain diagnostic value to the enlarged
clavicular glands. To be sure, Troisier called attention to the fact
intumescence of the supraclavicular glands is not specific to cancer
stomach, but is peculiar to all visceral cancers. Tarchetti considers
relling of the left supraclavicular glands as pretty characteristic. The
s are voluminous, easily palpable, sometimes visible, prominent
ly above the clavicle under the skin. On the other hand, Soupault
abbé call attention to the fact that palpable clavicular glands accom-
ng gastric cancer do not always speak for carcinomatous metastases.
various cases of examination neoplasmatic formations were not
l, but tuberculous products. As the enlargement of the supraclavi-
glands, quite aside from its rare occurrence, is undoubtedly a very
ymptom, it will but exceptionally enable us to take it in account as
isive diagnostic factor.

3. *Œdema Fugax.*

Even in the beginning of carcinoma, œdema of the ankles is seen occa-
ly and transitorily. I observed it in 12 per cent. of my cases. I
ler the fugacious œdema, if other factors causing œdema are absent, as
inestimable symptom of intestinal carcinoma. To be sure, such an
a may occur also in non-malignant gastric affection (I saw such a case
nsultation), and is, therefore, if present at all, likewise but a sustain-
ymptom.

4. *Condition of the Urine.*

There are essentially three kinds of anomalies of the urine aiding the
osis of gastric or intestinal cancer, namely: (1) the condition of
gen, (2) indigo-pigments, (3) presence of albumoses. (See page 253.)

(c) *Course and Duration of Cancer.*

The course of cancer of the stomach is characterised in the beginning
mple dyspeptic symptoms (pressure, fulness, anorexia, vomiting).

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wald. Klinik der Verdauungskrankheiten. II. 3 Aufl. P. 330.

Riegel. Krankheiten des Magens. 1897. P. 29 and 765.

Delbet. Société de chirurgie. January, 1900.

roisier. Arch. générales de médecine. 1889.

oupault and Labbé. Bull. de la Société médic. des Hôpitaux. July, 1899.

épine. Deutsche medicinische Wochenschrift. 1894. No. 13.

archetti. Deutsches Archiv für klinische Medicine. 1900. Bd. 67, P. 574.

Yet even these initial symptoms may either be missing, or may be only slight. The "red thread" in the clinical picture is and remains marasmus. Later the above-mentioned attacks of pain and vomiting are added. As ignis fatuus in diagnosis there occur temporary improvements, even increase of weight; their psychic effect and the resulting comfort may seduce patient and physician into an unjustifiable optimism. Even under these conditions, the subjective troubles do not disappear entirely, the facies carcinomatosa remains, and even considerable gains of weight melt away quickly. Duration of the affection varies according to the location and expansion of the carcinomatous process. The course of pyloric cancers, on account of the mechanic obstruction resulting from them, is the shortest; cancers on the curvatures take a much slower course. It is rather awkward to make a positive statement about the duration, because the first subjectively not well-marked beginning of the affection has frequently existed much longer than the patient states. From the beginning of manifest symptoms until death, one year is probably the mean duration of the disease. The statement of some authors that the earlier in life a carcinoma develops, the more rapid its course, is valid for some cases, but by no means for all cases.

Death ensues in the majority of cases from marasmus or through formation of carcinomatous metastases in liver, portal vein system, peritoneum, omentum, lungs, etc., or finally from exhaustive hæmorrhages, perforative peritonitis, formations of fistulæ, septic inflammation of lungs and pleura. In a few cases fatal ending is caused by addition of fever, the character of which has recently been more closely investigated by Hampeln,[76] Devic and Chatin,[77] and Freudweiler.[78]

The Early Diagnosis of Gastric Cancer.

As long as a cure of carcinoma of the stomach by means of the knife was not obtainable, an urgent demand for the earliest possible diagnosis did not exist. Only since there has been a possibility of resecting the carcinomatous degenerated pylorus or even larger parts of the fundus, science was given the problem of recognising the affection at the earliest possible time.

This question could, as has been done by various writers, be simply answered by the confession that we do not know of any absolutely positive early symptom for cancer of the stomach. Although I acknowledge this fact entirely, the question is, according to my opinion, by no means definitely settled. For there are indeed some cases—though not many—in which, after an early diagnosis had been made, an operation was successfully performed, *i.e.*, a tumour had been extirpated. It may be expected that physicians, and particularly specialists with more extensive practice in early recognition of cancer even not clinically developed, will be enabled to recommend surgical intervention to the patient earlier than at the present time.

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[76] Hampeln, Zeitschrift für klin. Medicin, 1884, Bd. 8, P. 221.

[77] Devic and Chatin, Province médic., 1892, P. 445; Reference to Hayem and Lion, Maladies de l'estomac, Traité de Médicine de Brouardel et Gilbert, T. iv.

[78] Freudweiler, Deutsche Archiv für klin. Medicin, 1899, Bd. 64, S. 544.

The fact that this was successful but in a minority of cases must not deter us more than the experience that we succeed in perfectly curing tuberculosis of the lungs but in a small number of cases.

What does early diagnosis of gastric carcinoma mean? It has been said, if no tumour can be palpated. This is certainly incorrect; for cases with palpable movable tumours have often enough been operated radically. But this statement is correct in as far as the tumour grown to palpable size usually extends already over a large part of the gastric walls and infects not only the neighbouring but also the more distant glands.

The requirement stated by Czerny and Rindfleisch,[79] and later by Kraske,[80] to operate in cases of pyloric cancer only if a tumour is not yet palpable, has a certain justification in as far as the hope of finding many a cancer suitable for radical operation among the carcinomata not palpably developed is certainly greater than otherwise.

The possibility of great progress depends on two entirely different factors; first to get the patient as early as possible under observation. In this respect we have no influence, and that particularly not because nearly every carcinoma has a longer or shorter latent stage at which, although objectively diagnosis might have been made, the patients do not complain of such considerable disorders that they are willing to undergo a medical treatment of any length of time, quite apart from submitting to surgical interference. When grave symptoms have made their appearance, in the majority of cases the time for radical operation has passed. Perhaps also the unblessed optimism of many physicians who treat patients of this kind for weeks and months simply for "catarrh of the stomach" or "nervous dyspepsia," without regarding the possibility or probability of a threatening carcinoma, contributes to the delay of surgical operation. Penzoldt's[81] earnest admonition not to rest in the treatment of patients above forty years of age and suffering from troubles of the stomach before the diagnosis of cancer is either made or refuted, has not been sufficiently regarded.

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[79] Czerny and Rindfleisch, Festschrift für Theodor Billroth, Stuttgart, 1892.

[80] Kraske, Berliner klin. Wochenschrift, 1894, No. 20 and 21.

[81] Penzoldt, in Penzoldt-Stintzing Handbuch der speziellen Therapie, Bd. iv.

At the present time the chief point rests in a detailed clinical analysis of each individual case. It must be distinctly understood: *no factor alone is decisive, but all together converge to a distinct ensemble.*

Some points in question may be particularly emphasised:—

1. *Anamnesis.*—The existence of heredity is rather rare (8 to 12 per cent., according to various statistics); greater suspicion is excited through repeated observation of cancer in the patient's family. According to my observation, "family" cancer occurs in 11 per cent.

An exceedingly important hint for which we are indebted to v. Leube is the above-mentioned rash occurrence of cancer while in the best of health and in persons who had not been suffering from gastric disorders up to that time. Though there are also exceptions to this rule, they do not invalidate it.

Rarer, but if present, remarkable, are traumatic insults of a graver nature received a longer or shorter time previously (not too long, however), and I emphasise this fact in spite of Kuttner's[82] contrary statements. It requires no special emphasis that such a symptom must be considered clinically with great reserve.

2. *Subjective Symptoms.*—At an early stage there is not one absolutely characteristic symptom. The most suspicious is a permanent anorexia not relieved by diet or medicine, as well as a disgust to animal nourishment. But even in pronounced cases there are exceptions to this rule. The other symptoms not being distinguishable from those of chronic gastritis, pyloric stenosis and occasionally also nervous dyspepsia, have hardly a diagnostic value. As soon, however, as they appear with undoubted distinction, the stage of early diagnosis is passed.

3. *Objective Symptoms.*—A striking loss of weight, recognised by regular weighing, in a person of normal weight up to the present disease, is certainly one of the most important clinical symptoms. Frequently, however, we make the mistake of not considering enough the factor of artificial loss of weight through withdrawal of food and decrease of fats and carbohydrates. Only loss of weight with simultaneous marked loss of sufficient nourishment is taken.

The most important result—always tumour excepted—is given by careful and repeated examinations of the gastric contents. Above all, there is the grave impairment of the motor function and of the secretion of hydrochloric acid and enzymes, and, resulting from it, abnormal lactic acid fermentation. If this trial is positive and the remaining clinical course speaks for a malignant neoplasm, the diagnosis of cancer of the stomach will but exceptionally be a mistake.

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[82] Lindner-Kuttner, Die Chirurgie des Magens, Berlin, 1898.

Aiding symptoms of decidedly diagnostic value are, as already mentioned, added by the observation of small and repeated additions of blood or pus in the contents of the stomach. To be sure, these are frequent enough in late stages of the process, but occasionally they are observed early if looked for carefully. The finding of carcinomatous particles, as mentioned above, does not pertain to the early stage of the process; this refers also to œdema at the ankles, alteration of blood, etc.

Based upon the above-mentioned symptoms, diagnosis is still not made early enough, but certainly earlier than it was possible two decades ago. There is no doubt that surgery in the course of time will draw advantage from these results.

[In an editorial of The Therapeutic Gazette (January, 1907), H. A. Hare, speaking on early diagnosis of cancer of the stomach, refers to Mouillin's article in the Lancet, September 22, 1906. Hare introduces Mouillin saying that, when there is definite evidence that one particular organ is not doing its work as it should do, when there is no reason to be found why it does not, and when it is clear that the patient's health is beginning to suffer, he holds that exploration is no longer a roving commission. Hare gives Mouillin's quotation of Hemmeter to the effect that "the simple continuance of a chronic gastritis, or nervous dyspepsia, in spite of logical and scientific treatment, accompanied with progressive loss of body weight during three or four weeks, justifies the suspicion of latent gastric carcinoma." Mouillin concludes his article: "If you suspect the existence of cancer you must not wait."]

Diagnosis of Atypical Forms of Cancer of the Stomach.

Besides the typical forms of carcinoma of the stomach there are frequently irregular atypical forms. The irregularity may manifest itself in three directions; the abnormal age of the patient, the latency of the symptoms, and the duration.

As to age, it is known that cancer of the stomach may occur in youthful persons. Marc Mathieu,[83] in 1884, compiled thirty-two cases of cancer occurring in young patients. I saw a case in a youth seventeen years old, another case in a girl of twenty-four years. Diagnosis in such cases is complicated with great difficulties, particularly because in that period of life sarcoma of the intestinal organs occurs much more frequently and because the manifestation of cachexia may be absent.

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[83] M. Mathieu, Gaz. des hôp., 1884, No. 118 (Monography, Paris, 1884).

Latent carcinoma (carcinoma occultum) is not exceedingly rare. Cases of this kind were described by Chesnel,[84] Raymond,[85] Brodeur,[86] Ewald,[87] Hampeln,[88] Leichtenstern,[89] Goldscheider,[90] Lafourcade,[91] Lefèbre,[92] Aron,[93] Fischer,[94] Friedenwald and Kotaling,[95] and others. In some cases there were no gastric symptoms at all *intra vitam*.

[William Osler (Philadelphia Medical Journal, 1900, p. 245) reports autopsies of 150 consecutive cases of carcinoma, mentioning a group of 7 cases with no symptoms during life.]

In most cases there were diffuse infiltrating cancers (parietal cancers), in others the cancerous formations did not occupy the ostia. In three cases of Chesnel,[96] the disease proper was obscured by a simultaneous pregnancy. The fact that diagnosis of gastric cancer was not made *intra vitam* in any of the mentioned cases proves the difficulty of a correct explanation *intra vitam*.

Finally, the course of a cancer may differentiate itself from the normal duration through an exceedingly rapid development of the process (carcinoma acutum). Schweppe,[97] reporting from the Göttingen Klinik a case of acute cancer of the stomach ending fatally within twenty weeks, compiled twenty-one other cases of acute development of carcinoma from literature. A case which terminated fatally within about five weeks from the onset of the disease was reported above (page 575) by myself.

The situation is especially difficult if no tumour is present, but the ensemble, particularly cachexia, suggests a malignant affection; or, though a pyloric stenosis exists, its character cannot be demonstrated in any manner whatever; or, finally, though a tumour is palpable, the determination of its character is made very difficult.

For all these cases there remains a diagnostically important though energetic measure: exploratory laparotomy (page 589).

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[84] Chesnel, Etude clinique sur le cancer latent de l'estomac, Thèse de Paris, 1877.

[85] Raymond, Progrès médic., 1882, No. 52; 1883, No. 1.

[86] Brodeur, Progrès médic., 1883, No. 34.

[87] Ewald, Klinik der Verdauungskrankheiten, 1897, 3 Aufl., S. 325.

[88] Hampeln, Zur Symptomatologie occulter visceraler Carcinome, Zeitschrift für klin. Medicin, Bd. 8, S. 221.

[89] Leichtenstern, Deutsche med. Wochenschrift, 1889, No. 6.

[90] Goldscheider, Deutsche med. Wochenschrift, 1890, No. 40.

[91] Lafourcade, Bulletin de la Société anatom., February, 1891.

[92] Lefèbre, Bulletin de la Société anatom., February, 1891.

[93] Aron, Deutsche med. Wochenschrift, 1896, No. 35.

[94] Fischer, Wiener med. Wochenschrift, 1896, No. 35.

[95] Friedenwald and Kotaling, New York Medical Record, 1898.

[96] Chesnel, *l.c.*

[97] Schweppe, Ein Fall von Magencarcinom mit anscheinend sehr schmellen Verlaufe in folge von allgemeiner Carcinose. Inaug.-Dissert., Göttingen, 1890.

Diagnosis of Ulcus Carcinomatosum.

According to observations of Rokitanski and Dittrich, as well as more recently of Hauser,[98] there is no doubt left that atypical cell-proliferation may develop upon the basis of a benign ulcer, which thereafter may entirely assume the character of a gastric cancer. To be sure, such a metamorphosis is not a very frequent occurrence. [The most recent view, as expressed especially by many surgeons, is rather to the contrary, and it is believed by some that as many as 90 per cent. of carcinomata have their starting point in an ulcer (Rodman, Mayo, Mouillin).] In 1891, Kollmann[99] was enabled to report fourteen cases, to which since that time a considerable number of observations have been added.

Diagnosis of ulcus carcinomatosum is based upon the fact of progressive cachexia with tumour accompanied by normal or superabundant hydrochloric acid production. *Vice versa*, there may exist carcinoma ex ulcere in spite of absence of hydrochloric acid (Rosenheim,[100] Hammerschlag,[101] and finally hyperchlorhydria may occur in cases of carcinoma, the origin of which cannot be referred to an ulcer. Besides hyperchlorhydria, history and course speak for the development of a carcinoma ex ulcere. Usually, in contrast to genuine gastric cancers, it concerns persons who had been suffering from gastric pains for many years. If hæmatemesis or melæna has been found in the history, diagnosis becomes more probable.

Frequently I observed in cases of ulcus carcinomatosum grave and repeated bleedings, which erroneously suggested the existence of round ulcer. Great difficulties are also encountered in the differential diagnosis between gastric carcinoma ex ulcere and tumour-like thickening of an ulcer. In such cases the finding of metastases in the liver or other signs of cancerous formation (ascites, peritoneal carcinosis) may be decisive. It need not be mentioned that cancer may be present in spite of absence of such symptoms. Diagnostic embarrassment may also be caused in differentiating between carcinoma ex ulcere and hypertrophic stenosis of the pylorus. We must, therefore, draw the conclusion that a positive diagnosis of ulcus carcinomatosum cannot be made with certainty; the course and other clinical manifestations, of course, may be suggested more or less in favour of a carcinoma. According to my experience, diagnosis is entirely impossible if a tumour is absent, and I made several mistakes in this respect.

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[98] Hauser, Das chronische Magengeschwür., Leipzig, 1883. (Literature.)

[99] Kollmann, Berliner klinische Wochenschrift, 1891, No. 5 and 6.

[100] Rosenheim, Zeitschrift für klinische Medicin, 1890, Bd. 17, S. 116.

[101] Hammerschlag, Archiv für Verdauungskrankheiten, 1896, Bd. 2, P. 1.

Diagnostic Aids in Doubtful Cases of Cancer of the Stomach.

(1) *Examination in the Warm Bath.*

This method, recommended by Chlapowski, Lennhof, G. Lee, Schuster, and Berkhan, may in doubtful cases give better information as to the seat of a tumour than the usual palpation, because by this means the relaxation of the abdominal walls prevents the mistake of rigid abdominal walls. In a few cases I employed this method successfully. It need not be mentioned that this means is also ineffective in many a case.

(2) *Examination in Narcosis.*

Through narcosis we succeed occasionally in gaining clearer information about situation, size, movability, and adhesions of a tumour than by means of the usually applied palpation. But at the present time narcosis is rarely made use of for diagnostic purposes. It is rather possible to gain information about the condition of the tumour immediately before or during the surgical operation.

(3) *Optical Methods.*

The employment of gastrodiaphany as well as the Röntgen rays has up to the present time not been of much use for the pathology of the stomach (see p. 115 *et seq.*). Perhaps gastroscopy (p. 119 *et seq.*) may, in the future, play an important rôle if we should succeed in constructing an easily introducible and dirigible instrument. The present instruments of v. Mikulicz, Rosenheim, Rewidzoff, Kuttner and Kelling are partly too complicated for handling, and partly do not permit a convenient and sure inspection of the gastric cavity. Kelling, whose efforts for improvement deserve our highest appreciation, is, after v. Mikulicz, the only one who recently successfully used the gastroscope in diagnosing dubious cases of gastric cancer. It cannot be foretold how far these efforts will lead to a far-reaching employment, nor what importance the method will have for the early surgical operation of gastric cancer—*and this is the salient point of the whole question.*

(4) *Exploratory Laparotomy.*

Exploratory laparotomy is performed partly for the sake of diagnosis, partly for deciding the possibility of an operation intended for removal of a tumour. The last-named indication will hardly encounter any objection. Although the exploratory laparotomy proper, as a diagnostic or differential-diagnostic means, may be theoretically justified, it will hardly receive general introduction, in consideration of the dangers still existing, though they may be slight, as well as the remaining concomitant inconveniences.

Differential Diagnosis.

The following possibilities must be considered as to differential diagnosis: (*a*) if a tumour is palpable, whether the same belongs to the stomach or not; or whether the tumour is malignant; (*b*) if a tumour is not palpable and cachexia is present, whether a carcinoma is present and to which organ it belongs.

(*a*) *Palpable Tumour.*

It is not always easy to tell whether a tumour belongs to the stomach or to a neighbouring organ. We have already discussed the most important points in this regard (page 567).

In order to determine more surely whether the tumour in dispute belongs to a certain organ, Minkowski[102] suggested inflating stomach and intestine successively, the former by an effervescent mixture, the latter by introduction of water, and to pay strict attention to the behaviour of the tumour. The following results were obtained, which, though we have to repeat things we have discussed before, for the sake of lucid visibility we compile in the table on the next page.

Is the tumour a malignant neoplasm or not? In the latter case, we have to consider fibromes or lipomes of the stomach, thickenings, gallstones, perigastritic processes which mimic a tumour, and others. Not one of the above-mentioned diagnostic factors can prevent the physician from making mistakes; we have to deal here with the weighing of the factor pro and con, on the basis of which the critic diagnostician forms his judgment. The most difficult and at the same time practically most important differentiation is that between an inflammatory process, an ulcer or a malignant neoplasm, or perhaps between both. A disease of several years' duration, presence of free hydrochloric acid, absence of peripheral glandular swellings would speak for ulcer and against carcinoma. *Vice versa,* short duration of disease associated with marked emaciation, pronounced motor insufficiency, absence of hydrochloric acid with simultaneous excessive formation of lactic acid, glandular swelling, œdema at the ankles, repeated small gastric hæmorrhages would speak with greatest probability for a malignant neoplasm.

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[102] Minkowski, Berliner klin. Wochenschrift, 1888, No. 31.

Tumours.

	On Inflation of the Stomach.	*On Inflation of Intestine.*
1. Of the Stomach		
(*a*) Pylorus	Move to the right and downward.	All gastric tumours move plainly upward.
(*b*) Anterior gastric wall and greater curvature.	Are felt broader and less distinct in their circumference.	
(*c*) Lesser curvature.	Disappear entirely.	
2. Of the liver.	Move upwards and to the right; anterior edge of the organ becomes more distinctly palpable.	The lower border moves upward; a tumour of the gall-bladder is pushed forward. If the tumours are very large, displacement may not occur at all.
3. Of the spleen.	Move to the left, often also downward.	Move upward to the left. Movable tumours become palpable in the normal splenic region.
4. Of the colon.	Move downward.	Do not move upward.
5. Of the kidneys.		Move first a little upward, disappear finally in the depth. The movable kidneys return to the normal kidney region. If neoplasms of the kidney are large, the median border becomes indistinct.
6. Of the great omentum.	Move downward.	Move downward.
7. Of the pancreas.	Disappear.	

(*b*) *Tumour is Not Palpable.*

The differential diagnosis is to be considered in cases when progressive debility makes a malignant process possible. Diagnosis may be made still more difficult, because, as experience teaches, in a few cases carcinoma develops very slowly and often more or less without symptoms, so that neither progressive debility nor other symptoms of a serviceable kind may be present. If the diagnosis in such cases has been nevertheless made correctly, there is less cause to admire the skill of the physician than his luck.

These rare cases excepted, cancer of the stomach may be confused with all diseases associated with cachexia and distinguished by a certain deficiency of symptoms, such as Addison's disease, progressive pernicious anæmia, even tuberculosis of the lungs, which frequently (30 per cent.) complicates cancer of the stomach, of course, only then when the symptoms are not very well pronounced. To discuss this subject in detail does not belong to the sphere of this work.

We limit ourselves to discussing the main differential-diagnostic points in the gastric affections proper. According to my opinion, all varieties may have to be considered under certain circumstances: chronic gastritis, gastric ulcer, ectasia of the stomach, stenosing hypertrophy of the pylorus, certain neuroses of the stomach.

(*a*) Differential diagnosis between ulcus ventriculi and carcinoma, compare Chapter on Ulcus Ventriculi (page 444).

(*b*) Between carcinoma and chronic gastritis. (See page 417.)

(*c*) Between benign and carcinomatous pyloric stenosis.

In this respect there is the question about spastic, cicatricial, and hypertrophic stenosis of the pylorus. The differential diagnosis between these various forms draws attention in case of presence as well as absence of a palpable tumour. Here we can discuss only the latter case. Has an ulcer preceded, or is it still existing, or is there a carcinomatously degenerated ulcer, or an inflammatory pyloric hypertrophy, or, finally, have we to deal with a spastic obstruction of the pylorus? The last-named possibility is probably soon decided; the others, however, only on the base of the course and possibly long-extended and clinical observations. The finding of the gastric contents may be very deceptive. Hyperchlorhydria and total achlorhydria with lactic acid formation may be common to benign and malignant stenoses, the former, of course, more frequent in the benign cases, the latter in the carcinomatous cases. In these cases also we will have to pay much more attention to the repeated finding of occult gastric bleedings than has been done heretofore, though this also may not be a compelling criterion. As we have thoroughly discussed the individual points in the Chapter on Motor Insufficiency, as well as in the present chapter, repetition of the discussion may be omitted.

(*d*) There is no doubt that in a case of simple nervous dyspepsia, such a grave debility may occur that at certain stages of the affection we must think of a malignant process, especially if there is simultaneously absence of hydrochloric acid and ferments. There is not a single characteristic sign for the differentiation of these cases, but only a co-ordination and comparison of all concurring symptoms: age, duration of disease, general condition of health, condition of the entire nervous system; besides, there is the finding of gastric contents, which, when normal as to motor and secretory function, or rapidly alternating from inacid to superacid condition, would speak, with a few exceptions, in favour of a functional disturbance. But even if hydrochloric acid and ferments are absent, it is safe to exclude cancer when the disease has undoubtedly lasted a long time, unless there are present marked motor disturbances, which are always suspicious.

Treatment of Cancer of the Stomach.

(1) *Medical Treatment.*

The number of those who seek safety of treatment merely in the finding of specific remedies becomes to-day constantly smaller; we have learned—and we are indebted for it chiefly to the signal activity of v. Leyden—to value another side of treatment, aiming to support and to strengthen the patient in his struggle against the hostile forces. The better we are instructed about the nature of this work of destruction, the more effective weapons of defense can be found. For the time being, we are still far distant from this goal in the treatment of cancer of the stomach, but nevertheless we have at our disposal therapeutic curative and palliative remedies of an importance not to be undervalued. We know, through the excellent investigations of Fr. Müller and Klemperer, that the more albumen is introduced, the more is decomposed by the carcinomatously infected body. The attempt to inhibit the proteid decomposition by abundant introduction of proteids must therefore be regarded as illusory.

As a rule the same principles are valid for the nourishment of persons affected with carcinoma as for persons with general reduced nutritive conditions. We have, however, to deal with two great and partially insuperable difficulties: on the one hand, with the invincible disgust for the ingestion of food; on the other hand, with the fact that some kinds of food remain undissolved in the stomach, or, even if such is not the case, do not leave the stomach on account of obstruction of pylorus or even without this, or leave it partially, whereby they lead to abnormal decompositions with consequent vomiting.

These defective circumstances guide us to direct the feeding of carcinomatous patients according to the following points of view: only small but very nourishing substances must be administered. The food must be given in pulpy, or, even better, in fluid form, using the liquid form exclusively when the pyloric stenosis is of excessive degree. The foundation of the nourishment, as in other debilitated persons, will always be milk, because this allows the largest number of variations and additions. An addition of coffee, tea, cocoa, of various leguminous flours, or the replacing of sweet milk with buttermilk, sour milk, or kefyr may make the use of milk an enjoyable and beneficial one for the patient.

The same may be said of vegetable purée soups, which, by the addition of appropriate seasoning, may exert a stimulating effect upon the lost appetite. The most appropriate alcoholic liquors are Tokay, port wine, diluted cognac, and, if great prostration is present, even small doses of champagne. Fats of good quality, if pronounced fermentation is absent, are a valuable and necessary nutritive substance in cases of cancer. The best are good butter, [cream], cocoanut butter (distinguished through almost complete absence of fatty acids), and the so-called Hauswaldt vigor chocolate (Kraftschokolade v. Mering), which deserves attention on account of its considerable amount of fat having been brought to emulsion. Bouillon and meal soups, with addition of eggs and albumose preparations (the value of which, however, we do not rate very high), are likewise to be recommended. Fish meat might find greatest appreciation among animal food substances, while poultry, beef, and veal are usually rejected.

As a whole, one should not be too rigourous in the restriction of diet in cases of carcinoma; experience often shows that such patients bear most excellently many a food which otherwise would be hardly advisable, while other substances usually considered easily digestible cause troubles in spite of all expectations. Even for humane respects so-called refreshments (fruit, beer, preserves, etc.) should not be forbidden too rashly, as their allowance often favourably influences the psychic condition, which is closely associated with certain sensations dependent upon nervous influences, such as appetite, sleep, etc. Much depends on the skill, experience, and individualising talent of the physician, without which even the most special directions fail in their purpose. In many cases, especially when absorption is markedly reduced, nutritive enemas are, though not a sovereign remedy, a means to support the nutritive treatment, which in practice could not be very well renounced. On the composition of the nutritive enemas and the techniques of their application, see page 304 and the Chapter on Gastric Ulcer, page 455.

Besides diet, the patients should submit to the greatest mental and bodily restraint, as a rule, to be obtained easily, as on account of increasing debility they spontaneously desire much rest.

The further treatment must deal with the most striking symptoms, namely, anorexia, vomiting, pain, and their consecutive result, sleeplessness.

In regard to the first-named factor, cortex condurango enjoys such a widespread fame as a stomachic that it may appear heretical to touch it; but I must openly confess that condurango almost always fails in truly grave cases of anorexia. Nevertheless, we may not wish to be without it. It is used either as infusion, as vinous extract (vinum condurango), or as fluid extract. Since the latter, having been adopted into the German Pharmacopœia, has been warranted as a good and uniform preparation, I use it exclusively.

℞ Fluid extract condurango.......................... 50.0
Sig.: One teaspoonful in a wineglassful of water half an hour before meals three times a day.

As soon as inclination to diarrhœa exists, I combine fluid extract condurango with fluid extract colombo in equal parts, one teaspoonful three times a day. Tinctura chinæ composita, elixir aurantiorum, tinctura gentianæ, tinctura amara, and other similar mixtures serve as a means of change which the helpless patient and the perplexed physician feel as necessary.

An essentially better stomachic in cases of cancer of the gastric cavity associated with retention is the washing-out of the organ. Undoubtedly anorexia is conditioned to a great extent by stagnation in the stomach. At the same time lavage of the stomach is the best anti-emetic, and I usually recommend medicines only in such cases where lavage is entirely useless, as occurs once in a while.

The result of gastric lavage in cases of cancer is at first alluring: appetite increases, a feeling of euphoria exists, which makes the patients optimistic and excites hope that they escaped from the threatening sword of Damocles. Only too soon is it shown that the hope had been deceptive; pain and vomiting disappeared, but the bodily strength is gradually sinking; the sense of life, increased for a short time, gives place to hebetude and despair.

The favourable effect of lavage is frequently missed entirely. Repeatedly I experienced that patients with cancer of the stomach very quickly collapse after application of lavage, and feel better after its entire omission or after its very rare application. Under such conditions I cannot help emphasising the little effect of anti-emetics, as for instance, of resorcin, particularly in grave cases. When in such desperate cases we wish to count upon good effect—of course, only palliative—there is nothing left but replacing gastric digestion by systematic rectal feeding; but in cases of cancer we must not continue this feeding too long, at the utmost for three to five days, as, according to my experience, a rapid loss of strength usually ensues after rectal feeding.

If artificial feeding cannot be entertained on account of too great weakness, a quite specific dietetic regimen must be introduced. The food must consist of small quantities of easily absorbable substances, namely, cream, beef-tea, albumoses, gelatine, biscuits, meal-pap, ice-cold champagne, sherry, cognac, and similar liquors. When the stomach is very irritable, food must be given only at intervals of one hour.

Of medicines I prefer most chloroform (3 to 5 drops on ice), whereas chloroform water is of slighter use. Opiates (morphine, codeine) and all other narcotics are best avoided as long as possible; if they have become absolutely necessary, hypodermic or rectal administration is to be preferred to that by the mouth.

Pain may occur in such a varying degree in cases of cancer that we must consider partly no medicine at all, partly only weak sedatives, partly and finally, narcotics in effective doses.

If pain is but slight, a few drops of tinctura valeriana, Hofmann's anodyne, a mustard plaster, cold or hot packs may be sufficient; if the attacks are more severe, codeine with belladonna in suitable doses (codeine-phosphate 0.03, extract belladonna 0.01, sacchar. lactis 0.5; one or two powders in an attack) is effective; likewise suppositories of codeine (0.05) and belladonna (0.03) are very efficacious. In cases of severe cardialgia we can hardly do without morphine (hypodermically 0.02, and in cases of vomiting, with the addition of atropine 0.0003 to 0.0005).

Under some circumstances, particularly in cases of carcinomatous pyloric stenosis, obstinate *constipation* is a symptom against which some measures must be taken. In many cases enemas (if necessary, with addition of soap, castor-oil, codliver-oil, and sodium carbonate) may be satisfactory; in very obstinate cases rhubarb in powder or infusion, fluid extract of cascara sagrada, and salts may be recommended. In other cases diarrhœa may be prevalent; we use, besides colombo, the so-called intestinal antiseptics (salol, bismuth salicylate, bismuth, beta-naphthol or benzonaphthol, [iodomuth]). Here, also, if possible, I avoid an excessive administration of opiates.

(2) *Operative Treatment.*

Since Billroth, in 1878, first demonstrated the practicability of the radical operation for gastric cancer, the operative treatment for that condition has become the prevalent custom among clinicians and surgeons. Since then over a quarter of a century has elapsed, during which decided improvements have been made in the technique, much information has been gained as to the anatomic and pathologic relationships of gastric carcinoma, and unquestionable advances have been made in the diagnosis. Nevertheless many surgeons, and naturally even more clinicians, are still pessimistic in their views on the radical operation for gastric carcinoma; indeed, we still hear every now and then loud denunciations by physicians against radical operation in general, and they still recommend only palliative measures. These observations are, however, not altogether unjust, especially from the standpoint of direct results; for resection of the carcinomatous stomach is still followed by the loss of many lives. Thus Lindner[103] states that even now the mortality rate is still 50 per cent., and even so eminent an abdominal surgeon as v. Mikulicz[104] reported a mortality rate for 1896-1898 of 44 per cent., against 32 per cent. in the previous years. The results are far more favourable, on the other hand, when, as was recently shown by Terrier and Hartmann,[105] the statistics are based upon the total reports of a number of prominent abdominal surgeons (Czerny, Krönlein, Carle, Mikulicz, Kocher, Hartmann). Out of 127 operations collected thus, 33 resulted in death, that is, a mortality rate of 26 per cent. Considering that at the beginning of the era of resection, 20 patients died out of every 25,[106] it is indeed evident that the results of the radical operation, excepting slight deviations, are gradually becoming more favourable.

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[103] Lindner, Die Chirurgie des Magens, Berlin, 1898, S. 309.

A number of conditions are apparently accountable for this improvement: the greater ability to recognise the disease, which, however, has not yet reached its highest stage of perfection; the tendency for patients to favour earlier operations; the careful selection of cases; and finally, the perfected technique of the surgeons. The internists are as much divided in their opinion on this question as are the surgeons, and in reference to the selection of cases, it cannot be emphasised too frequently that it is the duty of every clinician to consider the advisability of operation in every case of gastric carcinoma. If he has had experience in this line he will soon be able to exclude the unsuitable cases, which, as a rule, predominate. The suitable cases, of which a certain percentage will still be excluded when laparotomy is performed, must be recommended to a surgeon without delay. In cases which are unsuited for resection, the operation may be postponed and the attempt made to relieve the symptoms by palliative treatment, diet, and other measures; but in cases favourable for operation, every day of medicinal treatment after the diagnosis has once been established means a serious procrastination. Unfortunately this standpoint is frequently fallacious on acount of the differences and the dissensions of the physicians in their observations, which are naturally transmitted to the patients and their relatives. The fate of many cases, suitable for operation, will be hastened, as long as patients will fail to regard the operation for cancer as a *prima* instead of as *ultima ratio*. Accordingly, depending upon a wide experience, we recommend a radical operation as soon and as long as it can be performed. Of course, in a strict and careful selection of cases the number suitable for resection will not be great, and may perhaps fall below the present percentage, but the mortality rate for resection will certainly sink when patients to be operated upon are sent to a surgeon immediately after the diagnosis has been determined. The question presents itself, then, in what way can the "suitable" cases be recognised? This question can be answered in the majority of cases, yet not always. To reach a decision we may proceed as follows: involvement or non-involvement of the pylorus is always a determinable point. If the site is doubtful, the case must *a priori* be rejected as regards the indications. If the pylorus is certainly involved (marked dilatation, symptoms of stenosis, large amount of residue, gastric rigidity), we must determine whether or not a palpable tumour is present. In case of the former, the mobility of the tumour and the absence of complications (ascites, tuberculosis, weakness, arteriosclerosis, fatty heart, etc.) will serve as guides. If no tumour is present, but the diagnosis is highly probable, the same principles hold true, excepting that we remain in the dark as to the size and nature of the tumour. A positive decision can only be reached by performing an exploratory laparotomy. Since, however, even under these circumstances a palliative operation—gastro-enterostomy—comes into consideration, we should also advise operation in such cases with the expectation that either one or another case be found suitable for resection.

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[104] v. Mikulicz-Kausch, in Handbuch der praktischen Chirurgie, 1900; Separatabdruck, S. 148.

[105] Terrier and Hartmann, Chirurgie de l'estomac, Paris.

[106] Braun, Ebstein-Schwalbe's Handbuch der praktischen Medicin, 1900, Bd. 4, S. 1116.

A few surgeons have recently even gone so far as to attempt by operative procedures to remove extensive carcinomatous involvement of the fundus. As we know, successful attempts at resection have been made to remove almost the entire stomach from dogs and cats (Czerny and Kaiser,[107] Carvalho and Pachon,[108] de Filippi,[109] Monari[110]). With these experiments in mind at first, Connor,[111] of Cincinnati, and later Maydl,[112] Langenbuch,[113] Schuchardt,[114] attempted extensive gastric resection, or, if one chooses to call it so, total resection of the stomach for carcinomatous infiltration of that organ; in the majority of these cases, however, small portions of the stomach were left behind, so that, literally, the term gastrectomy is somewhat exaggerated. Schlatter,[115] of Zurich, is credited with performing the first total and at the same time successful resection of the stomach. Brooks Brigham,[116] of San Francisco, and Richardson, of Boston, succeeded in accomplishing the same feat a few months later. Böckel,[117] of Strassburg, performed a total gastric resection with good results quite recently.

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[107] Czerny and Kaiser, Beiträge zur operativen Chirurgie, Stuttgart, 1878, S. 141.

[108] Carvalho and Pachon, Arch. de physiol. normale et pathol., January, 1894.

[109] de Filippi, Deutsche medicinische Wochenschrift, 1894, No. 40.

[110] Monari, Beiträge zur klinischen Chirurgie, 1896, Bd. 16, S. 479.

[111] Connor, Medical News, 1884 (quoted from Terrier and Hartmann).

[112] Porges, Wien. klin. Wochenschrift, February 12, 1892.

[113] Langenbuch, Deutsche med. Wochenschrift, 1894, No. 52.

[114] Schuchardt, Archiv für klin. Chirur., 1898, Bd. 57, S. 454.

[115] Schlatter, Beitr. z. klin. Chir., 1897, Bd. 19, S. 757.

[116] Brooks Brigham, Boston Medical and Surgical Journal, May 5, 1898 (quoted from Terrier and Hartmann).

[Another case of successful total removal of the stomach was reported by G. Childs MacDonald,[118] San Francisco, September, 1898. Brigham's case was still doing well in February, 1900, when he reported a good condition of the patient, adding a photograph taken January 10, 1900, and mentioning the meal which the patient took on January 14, without the least trouble; a bowel of soup, a plate of chicken, with mashed potatoes and green peas, Italian bread and butter, and a cup of black coffee. In the spring of 1899 her weight was 110 pounds; January, 1900, 113 pounds.

Here also we cannot help mentioning the Rochester Dioscuri, William J. and Charles H. Mayo. In an article, "Radical Operations for the Cure of Cancer of the Pyloric End of the Stomach," Annals of Surgery, March, 1904, they report 41 radical operations after a method which they call a composite one. Thirty-seven operations were for cancer, 4 for inveterate ulcer. Of these, 13 operations have been made essentially according to their plan, with 1 death. There were 6 deaths in the remaining 28 cases, performed by various methods. In the last 11 cases their technique was followed throughout, and there were no deaths. (The mortality would be 17 per cent.) In recapitulation, Mayo gives the six important stages to the operation as follows:—

Step 1.—Open the abdomen. *Step 2.*—Double ligate and divide the gastric artery; ligate and divide the necessary amount of gastro-hepatic omentum close to the liver, leaving most of its structure attached to the stomach; double ligate and divide the superior pyloric artery and free the upper inch or more of the duodenum. *Step 3.*—With the fingers as a guide underneath the pylorus, in the lesser cavity of the peritoneum, ligate the right gastro-epiploic or gastro-duodenal artery, and progressively tie and cut away the gastro-colic omentum distal to the glands and vessels up to the appropriate point on the greater curvature, and here ligate the left gastro-epiploic vessels. *Step 4.*—Double clamp the duodenum, divide between with the cautery, leaving one-fourth inch projection; with a running suture of catgut through the seared stump, the end of the duodenum is closed as the clamp is removed; a purse-string suture about the duodenum enables the stump to be inverted; the proximal end of the stomach is double clamped along the Mikulicz-Hartmann line and divided with the cautery, leaving one-fourth inch projection; suture through the seared stump with a catgut button-hole suture; this is again turned in, after removal of the clamp, by a continuous silk or Cushing suture. *Step 5.*—Independent gastro-jejunostomy. *Step 6.*—Closure of the wound.

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[117] Böckel, Münchener medicinische Wochenschrift, 1901, No. 8, S. 320.

[118] MacDonald, Journal of the American Medical Association, September 3, 1898.

The after-treatment is simple—the head and shoulders of the patient are raised by four or five pillows, rectal alimentation is instituted, hot water by mouth after twelve hours in tablespoonful doses, increased to an ounce every hour. After thirty-six hours the usual experimentation with liquid foods is begun.

In a previous contribution on this subject (Annals of Surgery, July, 1903), Mayo recommended a somewhat similar operation, only that it was far more extensive, removing all of the stomach excepting the dome. Mayo continues: "With increased observation and experience, he feels the former operation, with a mortality of 3 deaths in 8 cases, to be unnecessarily severe for the average case of pyloric cancer. The operation described at that time has a place in surgery, and should be used in the cases of more extensive disease involving the body of the stomach. In these cases it has practically all the advantages of complete removal of the stomach, and should be used as a substitute for total gastrectomy where possible. The operation last described, with a mortality of 1 in 13, should be the operation of choice for the average of fairly early disease of the pyloric region."]

It may thus be concluded from these successful attempts that a total, or nearly total, removal of the stomach is technically possible, that the patients recover, increase in weight, and are able to thrive on a carefully selected diet. [*Vide* above, MacDonald's case.] Whether, however, these severe procedures will increase in popularity with further experience remains to be seen. It is also a question whether the duration of life is long enough to warrant the risks of so heroic an operation; for experience has taught us that when extensive infiltrating carcinoma exists, widely-spread metastasis is also already present. Accordingly in such cases, which are probably the most frequent, such dangerous operative measures are to be avoided. v. Mikulicz and Kausch[119] also hesitate in employing total resection of the stomach in such cases.

If we now leave this subject of total resection, which is still in its developmental stage, and return to resection of the pylorus, a question of deep interest to internists is suggested, namely: how do the patients thrive after resection of the pylorus, and how long do they live?

With reference to the first question, much knowledge concerning the functional ability of the stomach has been obtained through the investigations of Obalinski and Jaworski, Känsche, Rosenheim, Imredy, Mintz, Zawadski and Solmann, Maresch, Carle and Fantino, A. Mathieu, and

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[119] v. Mikulicz and Kausch, *l.c.*

Thiers.[120] These various investigators all proved that the motor activity of the stomach is decidedly improved after resection of the pylorus, and, according to v. Mikulicz and Kausch, as well as Terrier and Hartmann, it even surpasses the normal condition.

It has not yet been determined, after removal of the pyloric sphincter, to what extent a sphincter-like closure develops. I have demonstrated the constant presence of smaller or greater quantities of bile in the fasting stomach several months after removal of the pylorus, which tends to show that a sphincter shutting off the duodenum was not present. The secretory activity of the stomach is not altered in the majority of cases after resection of the pylorus, yet, as has been alleged by Rosenheim and Thiers, Terrier and Hartmann, as well as by v. Mikulicz and Kausch, and as I have confirmed, it is occasionally possible for the secretion of hydrochloric acid to again become established.

Lactic acid disappears with the removal of the stagnation; the statement of v. Mikulicz and Kausch, that lactic acid, even though in small quantities, persists in normal or increased motility without showing any evidence of lactic acid fermentation microscopically, is astonishing to me. As regards the duration of life after resection of the pylorus, we may again note that the results will become more favourable with the better selection of cases. The number of cures which last over three years* has indeed become notable in recent years, even though the average length of life of one and a half years has not increased. If the length of life in a certain case cannot be foretold, we should not, in my opinion, deprive the patient of the chance of a fairly long life, bearing in mind the average duration, provided no other contra-indication exists against the extirpation of the growth.

Since resection of the pylorus can unfortunately only be performed in a very small fraction of cases with the expectation of good results, gastro-enterostomy (gastro-jejunostomy) is still the operation to be preferred (see p. 528). Inasmuch as this is only a palliative measure, we must demand greater curative results than in case of the radical operation of pyloric resection.

What, then, is the status of the results? According to the last large collection of statistics by Chlumsky,[121] which included 500 cases of gastro-enterostomy performed from 1881 to 1896, out of 316 operated upon for malignant tumours, 160 died, 52.5 per cent. These results are not very encouraging, yet the mortality rate, according to Chlumsky, shows an unmistakable decrease. In 1881 to 1885 the rate was 75 per cent.; from 1886 to 1890, 48.4 per cent.; from 1890 to 1896, only 36.61 per cent. The latest results of v. Mikulicz present a mortality rate of 28 per cent., yet this percentage is decidedly less than that representing the loss of life following resection, and Lindner[122] has only a mortality of 11 per cent., in spite of his small amount of material.

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* I myself have a case in which the patient has now lived for five years since resection of the pylorus, and is enjoying perfect health.

[120] Cited from Terrier and Hartmann.

[121] Chlumsky, Beiträge zur klinischen Chirurgie, 1898, Bd. 20, S. 231.

[Mayo (Annals of Surgery, November, 1905) reports 421 cases of gastro-jejunostomy, of which 307 cases were benign, with 19 deaths (6½ per cent.). In the last 140 of the 307 benign cases were 4 deaths, a mortality of $2^6/_7$ per cent.; the last 80 cases gave but 1 death. One hundred and fourteen cases were malignant, with 21 deaths, a mortality of 18 per cent. Of these 114 cases, 63 were in connection with pylorectomy and partial gastrectomy, with 8 deaths (13 per cent.). The very unfavourable cases of cancer obstruction were subjected to gastro-enterostomy, so that this operation gives a higher mortality than radical excision. In the last 40 gastro-jejunostomies for malignant disease the mortality was 8 per cent. In the 421 jejunostomies there were 21 reoperated cases (5 per cent.).]

The length of the life of the patients operated on varies more or less with the operator. Billroth's patients lived and enjoyed health for one to eight months. The terminal results of Czerny and Rindfleisch[123] varied from twenty-five days to eleven and a half months. In general, however, the majority of the authors give an average duration of life of five to six months. However, several noteworthy exceptions to this rule exist. Thus Czerny,[124] Kappeler,[125] Alsberg,[126] Steudel,[127] and more recently H. Strauss,[128] have reported cases in which the patients lived for from two to five years after gastro-enterostomy. Strauss attributes this unusually long duration of life to the fact that the growths were but poorly supplied with connective tissue and cells. We must not neglect here to call also attention to the marked improvement in the subjective and objective symptoms and the noticeable increase in weight, which has been noted by nearly all surgeons. According to the investigations of Rydygier and Jaworski,[129] as well as Känsche[130] and others, the dynamic function of the stomach is markedly improved, whereas the secretory activity remains unchanged after this operation. On the other hand, we have seen not a few patients subjected to gastro-enterostomy who lived the average length of time, but whose health remained improved for a very short period of time only. Vomiting and pains reappeared shortly, ascites or icterus was soon superimposed upon the former symptoms, anorexia, which defied all treatment, developed rapidly and with it the strength soon failed. In a few cases the gastro-enteric fistula gradually failed to functionate, partly because the growing tumour constricted the fistula, and partly because a fresh carcinomatous eruption developed around the fistula with a similar effect.

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[122] Lindner, Berliner klinische Wochenschrift, 1901, No. 5.

[123] Czerny and Rindfleisch, *l.c.*

[124] Czerny, Berliner klinische Wochenschrift, 1897, No. 34, etc.

[125] Kappeler, Deutsche Zeitschrift für Chirurgie, 1898, Bd. 49, Heft 2 and 3.

[126] Alsberg, Münchener med. Wochenschrift, 1896, No. 50.

[127] Steudel, Archiv für klinische Chirurgie, 1898, Bd. 57, S. 454.

[128] Strauss, Berliner klinische Wochenschrift, 1901, No. 10.

[129] Rydygier and Jaworski, Deutsche medicinische Wochenschrift, 1889, No. 14.

[130] Känsche, *ibid.*, 1892, No. 49.

The conclusions as to the immediate results of the operation, the prolongation of life and finally the functional results, in all of which internists are interested, may be formulated as follows: gastro-enterostomy, in the hands of an abdominal surgeon who is certain of his technique, may be considered an operation of comparatively little danger, which assures the patient relief from his symptoms for a period of time lasting on an average of six months. Whether or not the patient should be advised to submit to this operation depends wholly upon the nature of each individual case. Cases in which the cancer has caused only a slight disturbance, or such as can be influenced by appropriate internal remedies, should of course be excluded. Belonging to this class of cases, as a rule, are those in which the carcinoma is located at the lesser curvature, or the rarer cases in which the tumour lies at the fundus or on the anterior or posterior wall. Even in carcinoma of the pylorus, the mechanical disturbances are not always so pronounced and the pains and vomiting not so resistive to internal medication that the establishment of a gastro-enteric fistula should be desired, or even be considered necessary. [In consideration of better surgical technique, these conclusions might be better modified.] Well-advanced marasmus also constitutes a contra-indication to the operation. Furthermore, the anterior gastric wall must not be involved by the carcinomatous process, for otherwise the difficulty of establishing a fistula would be greatly enhanced, if not impossible. Finally, gastro-enterostomy can not, as a rule, be performed in a small undescended stomach, whose walls are attacked by cancer.

Finally, we must take into consideration a method whose justification is yet to be proven, namely, jejunostomy. Maydl is credited with introducing this operation into surgery, with the object of using it especially in cases in which the larger portion of the stomach is destroyed by ulceration or new growth, and in which the stomach is no longer capable of insuring sufficient nourishment to the body. More recently the above-mentioned author proposed, basing his conclusions on a comparatively small amount of material to be sure, that jejunostomy be substituted for gastro-enterostomy, because this operation is less severe and also because it fulfils the important indications of relieving the stomach and preventing the new growth from being irritated. Maydl[131] reported 22 cases of jejunostomy with only 4 deaths, which is certainly a noteworthy result, although, on the other hand, less encouraging is the fact that of the 13 patients who lived longer than four weeks, only 7 outlived the third month and 1 the first year.

The permanent results are, accordingly, not near as lasting as those of gastro-enterostomy.

It must, therefore, remain uncertain whether gastro-enterostomy will be replaced by jejunostomy, although the comparatively simple technique and therefore direct results of the latter are in many respects alluring.

Appendix.

Sarcoma of the Stomach.

Besides the carcinomata, there are only the sarcomata among the tumours which incite a certain clinical interest; the other connective tissue tumours are practically so infrequent that their discussion, as far as diagnosis and treatment are to be considered, is hardly worth while. The sarcomata of the stomach may develop primarily and secondarily; the latter types, with the exception of the singular lymphosarcomata, are rare, though they are surpassed in this respect by the secondary cancers of the stomach.

The primary gastric sarcomata develop, according to Schlesinger,[132] in the shape of circumscript nodular tumours, usually located at the greater curvature, while the lymphosarcomata exhibit rather the character of flat infiltrations. The origin of the sarcomata is either in the muscular or submucous layer, while the mucous membrane itself usually remains intact. Only later the mucous membrane may be impaired by the permigration of the tumour toward the gastric cavity.

Gastric sarcomata occur frequently as mixed tumours, such as myosarcoma, myxosarcoma, fibrosarcoma, spindle-cell sarcoma, and angiocarcinoma. Their size may vary to a very great extent; Brodowski[133] observed a myosarcoma of twelve pounds.

As to age, there is, according to Schlesinger's statistics, no special age disposition to lymphosarcomata; but the second and third decades seem to be more disposed. Other sarcomata seem to prevail at a more advanced age, but, as in sarcoma, even the earliest childhood yields a considerable contingent. Sex does not show any particular difference as to frequency of its occurrence.

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[131] Maydl, Mittheillungen aus den Grenzgebieten, 1898, Bd. 3, S. 532.

[132] Schlesinger, Zeitschrift für klinische Medicin, 1897, Bd. 32, Supplementband.

[133] Brodowski, Virchow's Archiv, 1876, Bd. 67, P. 227.

[As to frequency of sarcoma of the stomach, it appears that it is more frequent than was formerly supposed. Fenwick says that whereas in 1897 Schlesinger was able to collect only 30 cases, in November, 1900, the number of recorded instances exceeded 60, of which 53 at least may be regarded as genuine. Although it is impossible to make any absolute statement as to relative frequency of the complaint, it is probable that the sarcomata constitute from 5 to 8 per cent. of all primary neoplasms of the stomach.]

Diagnosis and Differential Diagnosis.

Diagnosis of sarcoma of the stomach varies in but a few points from that of cancer. As regards *subjective* symptoms, they resemble almost entirely those of cancer. As in the latter, there are in the former also in the beginning obscure dyspeptic symptoms in the foreground; later vomiting, sometimes of coffee-ground-like masses, severe anorexia, and finally marasmus follow.

As to *objective* symptoms, there is likewise a striking analogy between the two varieties of tumour; a palpable tumour may be present or absent. If the tumour is situated at the pylorus, there develop the symptoms of stenosis. In cases of lymphosarcoma, however, there may be dilatation of the stomach without pyloric stenosis. The examination of the gastric contents, as far as known, likewise coincides essentially with that of cancer. Free hydrochloric acid is absent, if stagnation is present, there is lactic acid formation and development of lactic acid bacilli. [The sulphocyanide of potassium in the saliva gradually diminishes as the disease progresses, and finally disappears about one month before death (Fenwick[134]).] In all three cases observed by Schlesinger, blood was also found in the gastric contents.

The occurrence of particles of the tumour would be diagnostically decisive. A report of this kind has been made by Westpfalen.[135] Riegel[136] also mentions in his text-book on Diseases of the Stomach a case in which on hæmatemesis a large node of sarcoma was vomited.

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[134] [Fenwick, Cancer and Other Tumours of the Stomach, Philadelphia, 1903, P. 280.]

[135] Westpfalen, quoted by Friedr. Krüger, Die primären Bindegewebsgeschwülste des Magendarmskanals, Inaug.-Diss., Berlin, 1894.

[136] Riegel, Die Erkrankungen des Magens, II, P. 874.

According to Schlesinger, profuse diarrhœa is more frequently observed in cases of sarcoma than in those of carcinoma.

As may be seen, characteristic symptoms of sarcoma of the stomach are not present, and therefore great difficulties are encountered in the diagnosis differentiating it from cancer. Nevertheless, there are, as stated by Schlesinger, some hints which, under favourable circumstances, may allow the possibility of a differentiation. To these belong first the occurrence of metastases in the skin. By means of histologic examination of an excised particle of the skin, diagnosis may, under some circumstances, be made with great certainty. Of importance also is the enlargement of the spleen, which was found in all cases observed by Schlesinger, while, as is well known, it is but rarely met with in cancer of the stomach. Finally, according to Kundrat,[137] certain changes of the mucous membrane of the tongue occur in cases of lymphosarcoma; namely, enlargements and nodular appearance of the follicles, whereby the nodules, papillæ, and follicles show a quite particularly marked arrangement in the lines radiating symmetrically from the median line of the tongue towards the sides and to the front.

A positive differential diagnosis is, however, not permitted in spite of all these distinguishing signs; it should be remembered that v. Leube[138] found a true epithelial cancer of the stomach in a case of universal sarcomatosis of the skin. [Fenwick[139] mentions two other symptoms which may aid in the differentiation of sarcoma from carcinoma. "In many cases there is slight but continuous pyrexia, accompanied by rapid and profound anæmia, while in carcinoma fever is usually absent during the early stages of the complaint and the cachexia much more gradual in its development. Persistent albuminuria is often observed in sarcoma, but is exceptional in cancer."]

Treatment.

The treatment coincides entirely with that of gastric cancer, and is therefore a *surgical* one. There are some reports of relative cures of gastric sarcoma after extirpation in the early stage (Török, Salzer). Hopes for a longer preservation of life, however, should not be too exultant, as the disposition of sarcomata to recurrence is well known. [Schopf[140] reports a case, in which there was no recurrence at the end of a year, and Cantwell[141] reports the excision of a spindle-cell sarcoma weighing twelve pounds, whereby the patient got great relief for eight months.]

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[137] Kundrat, Wiener klinische Wochenschrift, 1893, No. 12.

[138] v. Leube, Spezielle Diagnose der inneren Krankheiten, 1 Aufl., P. 252.

[139] Fenwick, *l.c.*, P. 281 *et seq.* (There much literature.)

[140] Schopf, Centralblatt für Chirurgie, 1899, P. 1163.

The *medical* treatment does not vary in any particular from that of cancer. Schlesinger recommends the administration of arsenic (liquor kalii arsenicosi), under which treatment he observed sudden and unexpected retrogression of the neoplasma in cases of lymphosarcomata of other organs.

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[141] Cantwell, Annals of Surgery, 1899.

CHAPTER XXI.

Syphilis of the Stomach.

Preliminary Remarks.—Syphilitic affections of the stomach are exceedingly rare. Chiari[1] found among 243 autopsies of syphilitics only two cases of gastric syphilis. Stolper[2] also found among 86 autopsies of syphilitics only one case of lues of the stomach. E. Fränkel[3] likewise emphasises the rare occurrence of syphilitic ulcers in stomach and intestine.

In spite of the infrequency, gastric syphilis deserves a considerable clinical interest, so that a short discussion is indicated.

According to Mracek,[4] products of syphilis of the stomach may manifest themselves first as ulcerations of gummatous infiltration of the submucous layer; they occur especially in the region of the pylorus and at the lesser curvature, but also at the cardia; the infiltration expands towards the mucous membrane as well as towards the serous membrane of the stomach. Besides ulcerations, there are also gummatous products and scar-formations, so that we may conclude upon a possibility of cicatrisation of gummatous ulcers in the stomach.

Furthermore, there are, according to Mracek, ulcers resulting from syphilitic endarteritis of the gastric blood-vessels; they exhibit clinically as well as anatomically the character of the round ulcer of the stomach.

Frequently syphilitic changes of the intestines, skin, and liver, accompanying those of the stomach, have been observed.

Clinical important observations were made by Andral[5] as early as 1834, but recently the knowledge of gastric syphilis has been greatly advanced by the contributions of Fournier,[6] Dieulafoy,[7] and Max Einhorn.[8]

According to Einhorn, three groups of syphilitic affections of the stomach may be distinguished: gastric ulcers of syphilitic origin, syphilitic gastric tumours, and syphilitic pyloric stenosis. Einhorn contributed corresponding cases which are distinguished by the failure of the usual treatment, but by the rapid effect of antisyphilitic therapy.

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[1] Chiari, Festschrift für Rudolph Virchow, 1891, Bd. 2, P. 297.

[2] Stolper, Bibliotheca Medica, 1896, C. S. 6; quoted from Einhorn, Archiv für Verdauungskrankheiten, 1900, Bd. 6, P. 150.

[3] E. Fränkel, Virchow's Archiv, 1899, Bd. 155, P. 507.

[4] Mracek, Syphilis und venerische Krankheiten, Lehmann's Handatlanten, München, 1898.

[5] Andral, Clinique méd., tome IV, P. 121.

[6] Fournier, Communication à l'académie de médecine, January 18, 1898.

[7] Dieulafoy, Clinique médicale de l'Hôtel-Dieu de Paris, 1897-1898; Paris, 1899, P. 63 *et seq.*

[8] Max Einhorn, *l.c.*

Diagnosis and Differential Diagnosis.

The diagnosis of a syphilitic gastric affection is based upon the clinical examination as well as upon the demonstration of other syphilitic symptoms, and, finally, upon the effect of antisyphilitic medication.

The clinical picture as such, as far known at the present time, does not differ in anything from other non-specific gastric affections. Its importance is manifested essentially by the fact that its *course* differs in many respects from the usual. This fact refers especially to the syphilitic gastric ulcers which are characterised by unusual stubbornness, the uncommon disposition to grave gastric hæmorrhages, and the frequency of recurrence. If the anamnesis in such cases furnishes the fact of former syphilitic infection, if furthermore at the time of observation sure signs of the same are found, the supposition of a syphilitic affection obtains at least the stamp of great probability. If, finally, the gastric symptoms which up to that time appeared to be not influenced by the treatment used, disappear rapidly on administration of antisyphilitic medication, the suspicion of syphilitic origin grows almost to certainty.

We will, however, not omit to call attention to the fact that so competent an investigator as E. Fränkel[9] cautions against the view to regard all gastro-intestinal disturbances occurring in surely syphilitic patients as originating in specifically syphilitic alterations, even if the symptoms should disappear after administration of iodine and mercury.

The following very characteristic cases of syphilitic gastric ulcer may be mentioned:—

OBSERVATION I. Dieulafoy.[10]—Man, 33 years of age, admitted to the hospital with the classic symptoms of gastric ulcer, which had developed during one and a half years; treatment for ulcer (milk, kefyr, ice, morphine, cupping, points de feu); discharged without essential improvement. A few months later, readmission; the same subjective condition; ulcer treatment, alkalies, cupping in the epigastrium and at the vertebral column; no improvement. Eight months later, admission for the third time; gastric troubles increased; suddenly one evening severe hæmatemesis; milk-cure, alkalies, daily gastric lavage, tincture of iodine, vesicatories, points de feu. Although hysteric stigmata are absent, the idea of hysteric affection was suggested, and hydropathic cures of various kinds were employed. All these efforts were without effect.

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[9] E. Fränkel, *l.c.*

[10] Dieulafoy, *l.c.*

After another interval of eight months patient came again to the hospital in a wretched state of health; in the meantime there was another severe gastric hæmorrhage. Surgical interference was suggested. When he came under observation of Professor Dieulafoy all symptoms of a gastric ulcer were found, but at the same time also symptoms of a former syphilitic infection, which was conceded by the patient. After failure of a renewed short milk diet, patient received injections of green iodide of mercury dissolved in oil. After six days the gastric pains disappeared, patient could sleep, could take several litres of milk without pain. Four weeks later he had gained four kilogrammes in weight and was cured.

OBSERVATION II. Einhorn.[11]—Mrs. Carrie W., about 33 years old, had acquired syphilis seven years before. About two years ago she began to be troubled with her stomach, suffering usually from pains right after eating. Some time afterward a profuse hæmorrhage from the stomach occurred. According to the statements of the patient, she claimed to have vomited about a quart of blood mixed with food. She had then gone through a thorough course of treatment for the ulcer, and recovered, without, however, regaining her former state of health; for she complained almost constantly of dyspeptic symptoms, the appetite being considerably impaired, while frequent eructations and occasional gastralgia and constipation persisted. At the close of July, 1899, a second considerable hæmorrhage from the stomach ensued, and during two successive days the patient vomited large masses of blood, also passed blood in the stools (tar colour).

Status præsens: August, 1899, at the time of my visit, the patient is confined to bed and appears extremely pale and anæmic. Over the heart a slight murmur can be heard; otherwise nothing abnormal in the thoracic region. On palpation of the scrobiculus there is great sensitiveness to slight pressure; fever not present; neither sugar nor albumen in the urine. Patient complained of constant pain in the stomach, which radiates towards the back—and is increased even after fluid food—and of attacks of vomiting. She was placed upon a strictly fluid diet, absolute rest in bed enjoined, and bismuth, 2.0 grammes, three times daily, prescribed.

August 20, 1899, vomiting has ceased, and the pains are somewhat easier. The same treatment is continued during the following fourteen days, except that small quantities of semifluid food are permitted, and the patient is allowed to be up several hours during the day.

September 10, 1899, the condition of the patient has not changed since August 20th, and the pains persist in the same degree. In consideration of the history of syphilis, the possibility of luetic origin of the gastric ulcer appears feasible, and the patient now receives potassium iodide in increasing doses, the bismuth being discontinued. Several days after the administration of this drug the patient began to have less pains, and after the lapse of three weeks was completely free from any unpleasant sensations in the stomach. She was now able to tolerate ordinary diet without the least disturbances, gained in weight, and has since remained healthy.

[Simon Flexner[12] reports a case of perforating syphilitic ulcer of the stomach.]

Diagnosis and differential diagnosis may become much more difficult when a tumour is present. Relying on an observation in my practice, I particularly consider the differentiation from a malignant tumour as rather difficult, because carcinomata of the stomach and intestine, and especially rectal cancers, are not at all rare in former syphilitic persons, and temporary improvement of a striking kind may occur also in carcinoma. Only a long-continued improvement after antisyphilitic treatment, continued increase in weight, persistent disappearance of the tumour and of the manifestations of retention, if such were present, may be convincing as to diagnosis.

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[11] Einhorn, Philadelphia Medical Journal, February 3, 1900.

[12] S. Flexner, American Journal of the Medical Sciences, October, 1898.

Equal difficulties may be exhibited as to the diagnosis of a syphilitic stenosis. As we now know that intermittent (relative and spastic) stenoses occur and are repeatedly recurring, a stenosis of luetic origin must be deduced only with the greatest caution, under long-continued observation, and with critical consideration of all other factors.

[Hemmeter[13] recognises another form of pathological syphilitic changes in the stomach—the "diffuse syphilitic gastritis (chronic form)." This form, in the majority of cases, is probably due to what Chiari terms indirect syphilis—that is, due to circulatory disturbances, passive congestion, hæmorrhages, etc., produced by syphilitic diseases of other organs, especially of the liver. Among other more prominent symptoms in such cases, gastralgia may be mentioned. Allen A. Jones[14] has reported two cases of gastralgia, which were undoubtedly caused by syphilis. According to Hemmeter, chronic gastritis, due to syphilis, is a rather frequent and important syphilitic affection, and is one of the main causes of the poor state of nutrition in luetics. Histologically, it may be found to be a simple chronic gastritis, or else combined with gummata or gummatous ulcer. Syphilitic chronic gastritis, in the absence of gummata, does not differ pathologically from ordinary chronic gastritis, except in the greater frequency of small round-cell accumulation, especially in the submucosa, sometimes appearing like miliary gummata. Chronic syphilitic gastritis may develop from repeated attacks of the acute form, just as with non-specific gastritis. Relapses are not necessarily due to syphilis.

If characteristic syphilitic lesions exist in the liver, kidneys, spleen, pancreas, or intestines, the chronic gastritis should, in my opinion, be attributed to syphilis. In tertiary syphilis the remarkable malnutrition is due to a chronic luetic gastritis.

Hemmeter reports very interesting cases of two children of a syphilitic father, who were suffering from incessant vomiting, gastralgia, nausea, eructations, and who were cured by mercurial inunctions and saturated solution of iodide of potassium. In both of these children the vomit during the attacks contained no free HCl, but enormous quantities of mucus, and curdled milk but weakly. Both HCl and ferment secretion were restored by the treatment. Another case of syphilitic gastritis was observed by Hemmeter (autopsy made by Professor S. Flexner, Johns Hopkins Hospital) in a syphilitic negro, who had gummata in the following places: (1) Frontal bone, extending into the meninges and frontal cerebral convolutions; (2) one in the liver; (3) one in the spleen; (4) three in the mesenteric glands; (5) one in the testes and epididymis). Hemmeter gives a very interesting and excellent pathological report on this case, with a full-page illustration.]

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[13] Hemmeter, Diseases of the Stomach, Philadelphia, 1902, P. 596 *et seq.*

[14] Allen A. Jones, Philadelphia Medical Journal, vol. iii, P. 958.

Treatment.

The treatment of luetic gastric affections must consist, of course, in the employment of mercury and iodine. If the bodily strength is satisfactory, the combination of the two remedies is to be recommended. Several authors have stated that the iodine preparations are strikingly well borne in syphilis of the stomach in contrast to other affections. Instead of the administration of iodides by the mouth, iodides may be administered also by rectum, or idodipin may be injected hypodermically. [I have frequently administered iodalbacid in cases where I regarded an iodine preparation useful in gastric disturbances.] Apart from the specific treatment, diet must be regulated according to the variety of affection, especially in the beginning of the treatment.

CHAPTER XXII.

Tuberculosis of the Stomach.

In contrast to intestinal tuberculosis, which is found in about 70 to 85 per cent. of all who have died from pulmonary tuberculosis, tuberculosis of the stomach is an exceedingly rare affection. Simmonds[1] found in almost 2000 necropsies of tuberculous patients, gastric tuberculosis only in eight cases; a similar ratio is given also by Steiner and Neureuther,[2] Letulle,[3] and Plambeck.[4] According to the observations of O. Müller,[5] Hecker,[6] and Biedert,[7] tuberculosis of the stomach seems to be more frequent in childhood. The affection is met with much more frequently in men than in women. Letorey[8] observed among 19 collected cases, tuberculosis 16 times in men and only 3 times in women.

Three forms of tuberculosis of the stomach may be distinguished: the tuberculous granulomata, the rarest form; secondly, miliary tuberculosis of the gastric mucous membrane, the most frequent form according to the investigations of Wilms[9] and Simmonds; and finally, tuberculous ulcer of the stomach. While the two first-mentioned forms usually have a latent course, the tuberculous gastric ulcer, of course only very rare, may exhibit distinct symptoms.

The usual situation of the tuberculous gastric ulcer is at the pylorus, because, according to statements made by Virchow and Rokitansky, the pylorus, in contrast to the fundus, possesses numerous lymph follicles, although there are some observations of tuberculosis of the fundus. [Tubercle bacilli were found in a gastric ulcer by Coats,[10] Mathieu and Rémond,[11] Musser,[12] and Serafini.[13] While the ulcers are usually single,

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[1] Simmonds, Münchener med. Wochenschrift, 1900, No. 10.

[2] Steiner and Neureuther, Œsterreich. Jahrbuch für Pædiatrie, 71, P. 105.

[3] Letulle, refer. Centralblatt für pathol. Anatomie, 1893, P. 760.

[4] Plambeck, Inaug.-Diss., Kiel, 1885.

[5] O. Müller, Münchener med. Wochenschrift, 1889.

[6] Hecker, *ibid.*, 1894.

[7] Biedert, Jahrbuch für Kinderheilkunde, Bd. 21, P. 158.

[8] Letorey, Contribution à l'étude des ulcerations tuberculeuses de l'estomac, Thèse de Paris, 1895.

[9] Wilms, Centralblatt für pathol. Anatomie, 1897, P. 783.

[10] Coats, Glasgow Medical Journal, 1886.

[11] Mathieu and Rémond, in Letorey's Thèse de Paris.

[12] Musser, Philadelphia Hospital Reports, 1890, No. 1.

[13] Serafini, Annal. clini. del. Osp. di Napoli, 1888.

Hemmeter[14] mentions a case from Professor Osler's clinic at the Johns Hopkins Hospital, in which there were numerous ulcers of various sizes. The stomach showed 118 to 120 areas of loss of substance over the entire organ, but most thickly on the anterior surface near the greater curvature. The sizes of the ulcers vary from a pin's head to 5 centimetres in diameter. Musser's case showed an ulcer 2.5 by 7.5 centimetres in extent.]

Gastric tuberculosis, as well as intestinal tuberculosis, must be referred, above all, to swallowing of sputa containing tubercle bacilli. According to Klebs[15] and Alice Hamilton,[16] small hæmorrhagic erosions constitute the portals of entry for the tubercle bacilli. Only in rare cases miliary tuberculosis and tuberculous gastric ulcers may occur by way of blood or lymph vessels (Wilms, Simmonds, Weigert, Kaufmann). [Habershon[17] assumes that infection of the gastric mucosa occurs in this way. While there is a disposition, says Hemmeter, for development of tuberculosis in the intestine, there are numerous cases reported where ulcer existed in the stomach, the intestines being wholly exempt. The deepest ulcers, when found multiple in the stomach, do not extend beyond the muscularis mucosæ, and the infiltration of the mucous membrane extends little further than the actual ulceration. These facts are very evident in the sections presented to Hemmeter by Dr. S. Flexner, and which were taken from the cases reported by Dr. Hamilton. Superficial small ulcerations still showed vestiges of glands, but without any recognisable distinction between oxyntic and chief cells. Tubercle bacilli were present in small numbers, both on the free surface of the ulcers and among the remnants of the glands.]

The clinical picture of gastric tuberculosis is very slightly characteristic. There may, to be sure, sometimes occur severe gastralgias and hæmatemesis, but these symptoms do not differ at all from those of the simple round gastric ulcer, which, as is well known, is also sometimes observed in patients suffering from tuberculosis (see page 430). Simmonds especially states that in the two cases where he had made autopsies in tuberculous patients with severe gastric symptoms, he had to deal with simple ulcers, and not with tuberculous ones. In several cases (Kundrat,[18]

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[14] Hemmeter, Diseases of the Stomach, Philadelphia, 1902, third edition, P. 592.

[15] Klebs, Tuberculose, Leop. Voss, 1894, P. 80.

[16] Alice Hamilton, Johns Hopkins Hospital Bulletin, April, 1897; quoted by Hemmeter, Diseases of the Stomach, third edition, 1902, P. 593; there exhaustive bibliography.

[17] Habershon, S. H., Trans. Pathol. Society, London, Vol. xlv, P. 73 (quoted by Hemmeter).

[18] Kundrat, Wiederhofer in Gerhardt's Handbuch, P. 443 *et seq.*

Paulicki,[19] Struppler[20]) fatal perforation occurred in the course of tuberculous gastric ulcers. Marfan[21] reports 6 perforations among 14 observations. Fatal hæmorrhages after erosions of a gastric artery have also been observed (Kundrat, Bignon). A tuberculous pyloric stenosis does not appear to have been observed, although we know of intestinal tuberculosis that the ulcers possess a certain tendency to cicatrisation.

Diagnosis.

Diagnosis of tuberculous gastric ulcer is almost without exception made only by necropsy. According to Struppler, there may be the possibility of a probable diagnosis in cases in which in advanced pulmonary phthisis, pronounced gastric symptoms, and hæmatemesis make a sudden appearance with paroxysms of severe pains and tympany of the epigastric region, which is exacerbated at the slightest touch. According to the above-mentioned cases reported by Simmonds, we must be very cautious in giving an opinion about the variety of the ulcer—if diagnostically, we come at all that far.

Recently Petruschky[22] has called attention to the possibility of making a diagnosis by injections of tuberculin. The reliability of the method, however, is very much doubted by competent authors (Simmonds, E. Fränkel,[23] Struppler), even partly by Petruschky himself.

As a rule, we will have to content ourselves with thinking of the presence of a tuberculous process in tuberculous patients who exhibit symptoms of a gastric ulcer, and particularly of hæmatemesis. Beyond this, any possibility of a scientifically founded decision is missing.

Treatment.

The treatment of tuberculous gastric ulcer in its acute or chronic stage does not deviate in any respect from that of the simple round ulcer of the stomach. Specific remedies are as rare as for tuberculosis of the lungs.

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[19] Paulicki, Berliner klinische Wochenschrift, 1867.

[20] Struppler, Zeitschrift für Tuberculose und Heilstättenwesen, 1900, Bd. 1, Heft 3 and 4.

[21] Marfan, Troubles et lésions gastriques dans la phthisie pulmonaire, Thèse de Paris, 1887.

[22] Petruschky, Deutsche medicinische Wochenschrift, 1899, No. 12.

[23] E. Fränkel, Münchener medicinische Wochenschrift, 1900, No. 7, P. 237.

[I cannot help mentioning a measure which may act in the way of prophylaxis. Every patient suffering from pulmonary tuberculosis ought to be warned against swallowing sputum. In several cases, I could stop a distressing diarrhœa in tuberculous patients by calling their attention to this negligence of swallowing sputa.]

CHAPTER XXIII.

The Nervous Diseases of the Stomach.[1]

Preliminary Remarks.—The gastro-enteric canal is closely connected with the branches of the vagus nerve, and the great sympathetic abdominal plexus is exposed to manifold disturbances of innervation. It is true, diseases based upon pathologic-anatomical changes (degeneration of Meissner's and Auerbach's plexus) has been observed so rarely that they can hardly claim any clinical interest, but functional neuroses are decidedly more frequent.

As a whole, the diagnosis of the neuroses of the stomach and intestines is difficult if we do not content ourselves with a probable diagnosis; but if we are striving for a thorough analysis, even in many cases it is not at all possible to distinguish whether a neurosis or a palpable affection is present. Such is, for instance, the case when general neurosis develops along with disturbances of digestion, or as a partial symptom of the latter. As in clinical medicine the maxim, "a potiori fit denominatio," prevails, diagnosis in such cases will be left essentially to the view of the diagnostician.

Frequently also combined neuroses are met with, for example, motor and sensory, or sensory and secretory neuroses. According to the prevalence of the one or the other symptom, this or that denomination will be preferred, or a special emphasis of the character of the neurosis is abandoned, and we speak plainly of nervous dyspepsia, or still more generally of "gastric neurosis."

The clinical picture is, furthermore, obscured by the circumstance that frequently the same secretory and motor disturbances which may be named as undoubtedly nervous, are also observed in organic gastro-enteric affections. Yet we possess a series of landmarks which serve us as useful hints for diagnosis. As these usually coincide in the majority of the gastric neuroses, we will, in order to avoid repetition, put them at the head of this chapter, with the limitation, however, that, as the symptom-complex is so variegated and their occurrence so irregular, as a matter of course numerous exceptions and deviations are presented.

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[1] *Literature.*—Compare, besides the text-books on Special Pathology and Therapy of the Diseases of the Stomach, the following monographs on Neuroses of the Stomach:—

Stiller, Die nervösen Magenkrankheiten, Stuttgart, 1884.

Oser, Die Neurosen des Magens, Wien and Leipzig, 1885.

M. Rosenthal, Magenneurosen und Magenkatarrh, Wien and Leipzig, 1886.

Jul. Glax, Ueber die Neurosen des Magens, Wien, 1887.

Handbuch der Neurasthenie, herausgegeben von Dr. Franz Carl Müller, Leipzig, 1893.

Soupoult, Les Dyspepsies nerveuses, Paris, 1893.

Binswanger, Pathologie u. Therapie der Neurasthenie, Jena, 1896.

Compare also the bibliography in the sections on the individual gastric neuroses.

The examinations of the gastric contents, as well as the physical exploration, are very frequently negative in neuroses of the stomach (see below). We have rather to put up with the anamnesis and the general impression which the patient makes. That hereby great deceptions may be intercurrent, we are taught every day. One of the chief causes of the mistakes rests, in my opinion, in the fact that we imagine in too sketch-like a manner organic and functional diseases as contrasts, and forget that just as organic lesions in the same organism may occur simultaneously, dependently or independently from one another, organic and functional changes may exist also. It is not saying too much if I state that there is no intestinal affection which does not influence, to a certain extent, sometimes greater, sometimes less, the nervous system in general, and the gastro-enteric nervous sphere in particular.

We must, therefore, carefully weigh the individual symptoms occurring in the clinical picture, in order to say: in this case there exists an organic disturbance, in the other a functional, and in the third a combination of the two.

1. The general nervous system exhibits changes in a more or less high degree. There may be present headache, insomnia, nausea, state of depression or excitement, in brief, increased impressionability. Of objective deviations there may be mentioned exaggeration and also diminution of reflexes of skin and tendons, hyperæsthesia in various regions, alternating with paræsthesia and anæsthesia in other regions; frequently there is constant or intermittent polyuria. The state of nutrition may be good, even flourishing, or (and this is the rule) may be reduced in various degrees.

2. The sphere of digestion is characterised by a state of "labile gastro-enteric function." This is manifested through subjective complaints, but also (and this is exceedingly characteristic) through the peculiar form of objective disturbances. The former are distinguished through the non-connection, or at least the loose connection, with the digestive act. Furthermore, the absence of a definite regular connection of digestive complaints with the quality and quantity of the ingesta is remarkable. If you question the patients in this respect, they usually answer that either spontaneously or on advice of the physician they kept pretty well dietetically since the beginning of their complaint. On further questioning, however, whether occasional indulgences were followed by increased troubles, they usually answer negatively. If in doubtful statements the

patients are allowed for a trial to return to their usual food for a few days, we learn that although they feel no better, they do not feel by any means worse than before.

This independence of the complaint from the digestive act stands, on the other hand, in marked contrast to the dependence upon certain extraneous, not always discernible influences. In many cases change of air and climate has an undoubtedly curative effect; *vice versa*, excitements as occurring in daily life may represent the inciting factors of deterioration. Mental overexertion, accompanied by consecutive irregularity of the normal time and duration for meals forms an important ætiological factor; in other cases disorders of the sexual sphere form the base upon which, if a disposition prevails, functional gastric neuroses may easily be established, and so forth.

These temporary variations in the general and local conditions of the objective symptoms are parallel to variations of objective symptoms. To these belong *the change of the secretory and motor functions of the stomach, perhaps also of the intestines.* The former is characterised by intense variations of the production of hydrochloric acid. Total inacidity may alternate in the most protean manner with quite normal hydrochloric acid secretion within short intervals, even within a day. I shall give some instances of this kind in the Section on Nervous Dyspepsia. The motor function changes likewise. Such is particularly the case in atony of the stomach, which, as mentioned above (page 483), very frequently occurs in neurosis of the stomach associated with general neurasthenia.

The intestinal function, too, seems to be subject to variations; the frequent alternation of constipation and diarrhœa, and the sudden occurrence of acute diarrhœa, are probably likewise due to certain influences dependent upon the nervous system.

Under these circumstances a classification of the separate gastric neuroses is a difficult and scarcely satisfactory task. Every attempt at classification and arrangement is more or less sketchy and arbitrary. It may be justified only in as far as thereby it might be possible to gain a better knowledge of some symptomatically more prominent neuroses.

We select as the dividing principle the one suggested by M. Rosenthal: sensory gastric neuroses, motor gastric neuroses, digestive (secretory) gastric neuroses, and vasomotor gastric neuroses. As the latter have, up to the present time, not been subjects of diagnosis and treatment, they may be omitted. The subdivision into irritative and depressive types, as suggested by some authors, seems to us to complicate still more unnecessarily the already difficult division.

I. Sensory Neuroses of the Stomach.

(1) *Bulimia and Acoria.*

Bulimia, or *cynorexia*, is the morbidly increased sensation of hunger; *acoria*, the absence of the normal sensation of satiation. Both conditions offer much akin; indeed, there may be transition from the one to the other.

(*A*) *Bulimia.*

Bulimia is partly an independent neurosis, partly a symptom of other organic affections. Leo,[2] to whom we are indebted for the best work on bulimia, observed this neurosis in cases of Graves's disease, of ulcus ventriculi with hyperchlorhydria, of chronic gastritis, of tape-worms, of diarrhœa, and of menorrhagia. Even carcinoma and ectasia ventriculi are said to be occasionally connected with the occurrence of bulimia. According to Fournier,[3] bulimia is said to occur also in luetics, most frequently between the third and sixth months of the disease, rarely earlier or later. Furthermore, bulimia is an accompanying symptom of hysterics, neurasthenia, hypochondria, psychosis, epilepsy, pregnancy, pulmonary tuberculosis, and diabetes. Occasionally there occurs morbid canine hunger in affections of the sexual organs in the male (Lallemand). Peyer[4] considers the occurrence as rare. Further on I shall report a case of bulimia in a patient suffering from sexual neurasthenia. We must, however, emphasise that polyphagia is not rarely mistaken for bulimia (see differential diagnosis). According to Rosenthal,[5] bulimia is more frequent in women than in men, at the time of puberty and between the twentieth and fortieth years. As diagnostically and differential-diagnostically objective methods of examination come under consideration only as far as they will yield the morbid foundation of bulimia, we must be satisfied with the description of the symptoms.

Diagnosis.

The most characteristic symptom for bulimia is the appearance of an impulsive spasmodic sensation of hunger which unconditionally demands satisfaction. During the attack the patient is seized by a state of exceedingly pronounced weakness (défaillance), becomes pale, and is hardly able to stand; indeed, he may actually faint when the desire for food and drink cannot be stilled as soon as possible. In this condition the patient forgets all ethical and social respects, he takes food wherever he can get it and of whatever kind it may be. With the ingestion of the first morsel or the first swallow (wine, milk, etc.) the paroxysm begins to disappear.

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[2] Leo, Deutsche medicinische Wochenschrift, 1889, No. 29 and 30.

[3] Fournier, Gazette hebdom., 1871, No. 1 to 3.

[4] Peyer, Ueber Magenaffektionen bei männlichen Genitalleiden. Volkmann's Sammlung klinischer Vorträge, No. 356.

[5] M. Rosenthal, Magenneurosen und Magenkatarrh, 1886, P. 6.

There occur deviations from this classic type, which was known to the Oriental nations and had been discussed as to its criminal aspects with greatest carefulness. The case manifests itself usually in such a manner that the patients, shortly after a regular meal, become hungry and feel compelled to eat, without a real "impulse" appearing in particular. Many a patient, as I have occasionally observed, feels the economic part of the bulimia the hardest, as it stands in the most vivid contrast to the finances at his disposition.

In other cases bulimia is "periodical," appears at intervals of a few weeks and disappears again; in still other cases a long period of bulimia alternates with one of anorexia.

According to Leo's investigations, some of these cases may be referred to a so-called supermotility, this means that the stomach empties itself at a time when normally it should be full; in some of my cases I could observe this factor.

For the sake of illustrating these conditions, and as reports of cases of this by no means too rare affection are not abundant, a few histories may be briefly recorded:—

C. K., house servant, 27 years. With exception of a bronchial catarrh lasting for six weeks while in the army, always well and had normal appetite. For the last two months sudden cynorexia and abnormal thirst. Amount of urine apparently not increased. Loss of weight, 10 pounds. Several furuncles. Bowels regular. Eructation, with heartburn and regurgitation of food. No particular weakness. Patient feels that he must eat all day; he eats more than double the quantity in comparison with former times. Lower border of the stomach two fingers' breadth below the umbilicus; there slight splash-sound. *No sugar in urine.*

Examination of stomach-contents: (*a*) Fasting stomach. About 10 cubic centimetres of a mucous fluid mixed with a few bread-remnants, and of intensely acid reaction. Total acidity, 2.1 per mille; intense hydrochloric acid reaction. (*b*) After test-breakfast; after an hour stomach empty (supermotility), filtrate exhibits intense HCl reaction; on repeated examination the amount of free HCl is 2.2 to 2.4 per mille. *Treatment:* Solutio arsenicosa Fowleri, 5 drops three times a day. After a period of two weeks bulimia has disappeared, but there is still occasional pyrosis; determination of HCl amounts to but 1.6 per mille.

In the following case, bulimia immediately followed puerperium:—

Mrs. A. G., 40 years of age, married eleven years, eight children, one of them dead. Last childbed sixteen months ago, normal. On the evening after childbirth, pain in abdomen and intense hunger appear for the first time. If hunger is not satisfied, patient faints. One or two hours after a heavy meal, again hungry. During sleep sensation of hunger ceases. Pain in the epigastrium appears only with appearance of sensation of hunger. Patient can bear all kinds of food. Menses regular. No sugar in urine.

The following case is evidently due to sexual neurasthenia:—

H. T., 49 years of age, farmer. For several years patient easily irritable, inclined to hypochondria. For the last year patient complains of peculiar digestive symptoms; he has a sensation "as if small animals were in the stomach," complains of belching, pain when moving around, and constipation. In the last three months patient, as soon as he starts to eat, is seized by uncontrollable hunger, compelling him to take a larger quantity of food immediately after a large meal. The desire for it is impulsive, so that he tries to procure food clandestinely. A few hours after eating, pressure and fulness in the epigastrium, which are relieved by motions. Patient never had a venereal infection, but suffered *from his eighteenth to twenty-fourth year from almost daily pollutions;* even at the present time there are very frequently pollutions. *Complete impotency for a year past.* Patellar reflex very marked, pupils react well, no Romberg's sign. Examination of the gastric contents shows free hydrochloric acid to the amount of 1.2 per mille. Otherwise no abnormality in the stomach. Urine contains no sugar or albumen.

Differential Diagnosis.

We must well distinguish between simple polyphagia in its different degrees and bulimia. As we consider as its most important and characteristic symptom the morbidly increased *impulse for food*, commanding satisfaction under any circumstances, all affections based merely upon increased appetite (as, for instance, in diabetes on account of increased oxidation in the body) have to be put aside. On the other hand, it must be conceded that in a malady manifested only through subjective symptoms the greatest latitude is given to the individual conception of the same.

(*B*) *Acoria.*

We mean by acoria (derived from α and κορέννυμι, I satiate) the *absence of the normal feeling of satiation.* Acoria cannot be made identical with polyphagia, as Rosenheim[6] does, nor with bulimia, for acoria may be associated even with anorexia. Peyer[7] rightly desires to differentiate between the latter and acoria. In some cases such a differentiation cannot always be maintained; other cases represent undoubtedly transitory forms.

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[6] Rosenheim, Pathologie und Therapie der Krankheiten der Speiseröhre und des Magens, 1891, P. 299.

[7] Peyer, *l.c.*, P. 26.

The essential symptom in acoria consists of the fact that the feeling regulating satiation, which is incited by a certain quantity of food, though it may vary in individual limits, is absent temporarily or constantly. Acoria is almost always secondary to general neurasthenia or hysteria. I observed four cases, two in men and two in women. The following two may be mentioned:—

1. G., teacher emeritus, 52 years of age, has complained for twenty-four years of gastric disturbances, which, according to his statement, began suddenly with stinging in the region of the liver and pressure in the stomach. Simultaneously loss of appetite appeared. *Appetite remained constantly bad.* There were pressure and "fulness," especially on an empty stomach; after eating, relief. Occasionally lightning-like pains in the gastric region; eructation and flatulency. The most disagreeable symptom for the patient is *the absence of feeling of satiety; "the stomach never tells when it is enough,"* and the patient must therefore measure the amount of his food according to his former experiences.

2. P., worker in metal, 28 years of age. Mother dyspeptic. Was well in childhood. According to his statements there appeared four years ago, after excitement, attacks of cardialgia with nausea and vomiting; the latter had finally a bile-like appearance. The attack lasted two or three days. These attacks ceased without medication. For the last two years sedentary life, with subsequent constipation, which is relieved only through the use of castor-oil or enemata. Appetite changing, occasionally absent. Patient, on medical advice, has taken 800 capsules of creosote for suspected beginning pulmonary tuberculosis. For the last three months complains of absence of feeling of satiety. A dinner does not give any satiation to the patient, although he eats twice as much as formerly; "one hour after dinner he can eat again." Patient is very much emaciated; mucous membranes pale, eyes deep in their sockets, pupils dilated, tongue furry, abdomen moderately distended, epigastrium not tender; greater curvature reaches down to the level of the navel; moderate splash-sound, no succussion. Kidneys not palpable; liver and spleen normal. Inspiratory position of diaphragm. Lungs show regular and uniform excursions; percussion sound at the left apex posteriorly somewhat deeper than at the right, respiration there a little harsh; otherwise nothing abnormal. Patellar reflexes increased, no Romberg's symptom, pupils react upon light and accommodation.

Peyer (*l.c.*) reports two cases of acoria in cases of pronounced sexual neurasthenia.

Diagnosis and Differential Diagnosis.

Diagnosis is given by the most important symptoms: absence of normal satiation and the demonstration of other neurasthenic symptoms. Occasionally, as already mentioned, there are transitions between acoria and bulimia, which perhaps may be explained by the fact that a hyperæsthesia of the gastric nerves may be followed by anæsthesia as a reaction. As to differential diagnosis between polyphagia and acoria, we have to consider that in the former the ingestion of an increased quantity of food is due to increased appetite or increased oxidation (diabetes), while in acoria the appetite may be either normal or (above Case I) even diminished. Acoria is differentiated from bulimia by the absence of the impulse, the morbid *eagerness for eating.*

[Hemmeter[8] reports an experience which, for ætiological purposes, deserves to be mentioned here. "As the feeling of satiation is absent after copious filling of the stomach with food, and as it cannot be disguised by an abnormal feeling of hunger, because hunger is normal or subnormal in acoria, another explanation that has been offered for this nervous affection is that it is due to loss of sensibility, or anæsthesia, of the gastric sensory nerves. This seems to be a very probable explanation, since we have personally had at least one experience that would suggest a local gastric anæsthesia as an explanation of acoria. The case is that of a young woman whose stomach we had sprayed with a three per cent. solution of cocaine and menthol. She returned on the same day, stating that, although she had taken a long bicycle ride after the spraying, and returned home feeling quite hungry, she had the impression that the food she ate never reached the stomach. She had no feeling in her stomach that the meal effected satiation. At first we overlooked the causal relation between the spraying with menthol and cocaine for this temporary acoria, and our attention was attracted to it after the same symptoms were complained of each time the menthol and cocaine were used. These agents had been employed for the relief of gastralgic pains resulting from erosions. It is conceivable that anæsthesia of the stomach-nerves may occur from repeated overdistension, as occurs in bulimia, polyphagia, diabetes mellitus, and dilatation of the stomach."]

Treatment.

The treatment for *bulimia,* when it is a secondary disease, must have regard for removal of the primary cause (genital affections, tape-worms, ulcer, etc.), or must combat any existing neurasthenia, hysteria, or psychosis through appropriate measures, which need not be discussed here, and finally must reduce the morbid irritability of the hunger centre itself. As suitable remedies we must mention first the bromides in small doses (0.5 to 1.0 pro dosi, two or three times a day). Furthermore, opium in combination with belladonna, as, for instance, the following formula:—

℞ Extracti opii,
Extracti belladonnæ .... aa 0.01
Sacchari 0.5
M. ft. pulvis.
D. tales doses No. xx.
Sig.: One powder morning and evening.

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[8] Hemmeter, Diseases of the Stomach, 1903, third edition, P. 809.

Or, according to Rosenthal, hypodermic injection of extractum opii; finally, arsenic (arsenious acid of 0.001 in granules or in form of Fowler's solution), such as

℞ Solutionis arsenicosæ Fowleri,
Aquæ menthæ piperitæ.............................aa 7.5
Sig.: Six drops three times a day, to be increased three drops every week.

Cocaine hydrochlorate (0.03-0.05—twice a day) also has been used successfully by Rosenthal. I have observed arsenic, in form of Fowler's solution, influencing most favourably numerous cases of bulimia. [Hemmeter saw in a coloured man, to whom he referred as champion glutton and who had subsequently developed bulimia, that the symptoms improved very much under the intragastric use of the constant current.]

Treatment for *acoria* can be successful only through removal of the general neurasthenia, namely, through hydropathic and electric treatment, climatic changes, etc. In a case of a hysteric woman in whom the absence of feeling of satiation was an element destroying her and her family's joy of living, I obtained an excellent and lasting effect through a Weir Mitchell rest-cure in the country. Of great importance seems to me in all neuroses, and particularly in acoria, a certain pedagogy in eating, whereby greatest attention must be paid to regulation of meal times, slow mastication, and to an accurately prescribed quantity of food.

(2) *Anorexia Nervosa.*

(Nervous Anorexia.)

Although the term "appetite" is apparently plain and clear, the differentiation of appetite from hunger is somewhat difficult. This much is certain, that appetite (desire for food) and hunger need not be combined. "A child often has appetite, when hunger is quite out of question" (Penzoldt[9]). Penzoldt very appropriately defines hunger as the admonition, appetite as the joy of eating. Loss of appetite is one of the most frequent symptoms of acute and chronic internal and external diseases, even of those in which the stomach itself is not involved primarily or secondarily. On the other hand, that anorexia is a frequent symptom in organic and functional digestive disorders is very well known.

By *nervous* anorexia is meant an entire absence of appetite, extending over weeks and even months, accompanied by direct repugnance or even disgust toward all food, though the digestive function may be intact.

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[9] Bibliothek der ges. medicinischen Wissenschaften, herausgegeben von Drasche; Article "Anorexia."

This condition is thought to be due to anæsthesia of the hunger center. The affection, found principally in women, is not a disease *sui generis*, but a more or less pronounced symptom acompanying hysteria, neurasthenia, or certain psychoses (melancholia, hypochondria, etc.). Peyer (*l.c.*) observed anorexia also in men suffering from sexual neuroses.

Anorexia may reach stages of different degrees, the highest approaching the absolute refusal of food by the insane (anorexie mentale). The course and prognosis depend upon the degree—fatal cases have not infrequently been observed—and prognosis must accordingly be made very cautiously. On the other hand, it is remarkable that patients with anorexia may maintain a strikingly good state of nutrition for months and years.

Diagnosis and Differential Diagnosis.

Diagnosis is easy as soon as nervous symptoms are manifested; more difficult, when such is not the case or some organic changes are concomitant. Furthermore, the picture may be obscured by simultaneous presence of organic diseases, such as gastric atony, displacement of kidney, liver, and other viscera. In all cases where the diagnosis "nervous anorexia" is to be made plausible, organic gastric affections, all of which *may* be accompanied by more or less marked loss of appetite, must be excluded. Above all, for instance, we will have to think of chronic gastritis. We are well acquainted with the stubborn loss of appetite in cancer of the stomach, which frequently appears as the first symptom, and consequently may make the diagnosis very difficult. It may be easily mistaken in incipient tuberculosis; if the physical and bacteriological examination is negative, the further course will bring a decision.

In the following we report a good paradigm of nervous anorexia:—

Mrs. E., 52 years of age, complains for two years of loss of appetite of a peculiar kind. There exists feeling of hunger, but no pleasure of eating. "The food does not want to go down." Patient can eat any kind of food with impunity. Patient has also lost the sensation of smell for some flowers, such as mignonette and violets; or these flowers rather have a disagreeable smell. Patient formerly underwent many excitements. No other diseases. Thirst normal, bowels slightly constipated. Climatic changes improve the patient. Examination shows perfectly normal conditions. Gastric contents likewise entirely normal. *Treatment:* orexinum basicum. After that sensation of hunger disappeared (?), no appetite present.

Treatment.

The treatment must fulfil several tasks; first, to ameliorate the general nervous symptoms; then, if necessary, to improve the state of nutrition; and finally, to combat anorexia itself. As to the former, the rich armamentary of measures and methods for neurasthenia and hysteria (hydro-electro therapeutics, climatic changes) has to be considered. Charcot[10] correctly advises isolating the patient from his family in order to facilitate a methodical change and regulation of diet and living. For this purpose, if the condition is due to anæmia, a regular *iron cure* would be recommendable. [Iron-tropon may be recommended in cases of this kind; it is an excellent drug-food, is exceedingly well borne; tropon by itself will provide plenty of nourishment in a small bulk, and can therefore be readily assimilated.] For the improvement of the state of nutrition a regular milk cure, where milk is tolerated, might be the most suitable; but we ought not to restrict ourselves to milk alone, but must add abundant carbohydrates and fats [Hauswaldt vigor chocolate], if, as frequently happens, an insuperable repugnance to meat and vegetables exists. In grave cases it will be necessary to the interest of the patient that he be fed artificially by the so-called gavage (page 303); in less severe cases, nutrient enemata may be sufficient.

In medicinal treatment, the so-called stomachica and amara might be tried in the beginning of the affliction; as a whole, and according to my experience, they will have but little effect. I wish to call attention to the decoction of cinchona bark (which in recent years has been unjustly pushed to the background) in the following formulæ:—

℞ Decocti corticis chinæ regiæ...................... 10.0 : 180.0
Acidii sulphuric diluti..........................gtt. 5.0
Syrupi zingiberis ....ad 200.0
M. D. Sig.: One tablespoonful half an hour before meals, three times a day.

Or:—

℞ Extracti fluidi cortic. chinæ.......................... 50.0
D. Sig.: One teaspoonful in a wineglassful of water before meals, three times a day.

Of similar effect is the above-mentioned (page 424) extractum chinæ Nanning, though it does not correspond by far to the high-sounding eulogies which are found in medical journals. In a few cases I saw good results from the extractum fluidum belæ indicæ (one teaspoonful in a wineglassful of water three times a day), as well as from orexinum tannicum (0.25 pro dosi). Dujardin-Beaumetz[11] recommends arsenic also in nervous anorexia.

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[10] Charcot, Œuvres complètes, Part I, P. 240.

Rosenthal observed favourable results from sodium bromide (0.5 to 1.0) before meals in cases where unpleasant feelings of pressure and fulness cause aversion to eating. In all cases of pronounced gastric neuroses, and especially nervous anorexia, we must warn against the use of mineral waters (iron waters excepted), which exercise a directly deteriorating influence in the majority of cases.

[A mixture which I have prescribed in quite a number of cases of deficient or lost appetite, and, as I believe, with much success, is as follows:

℞	(Acid. nitro-muriatici	15.0)
	Fluid extract. nuc. vomic	15.0
	Fluid extract. taraxaci,	
	Fluid extract. gentianæ......aa	30.0
	Glycerophosphates	60.0

M. D. S. One teaspoonful in one-fourth glassful of hot water half an hour before meals, three times a day.]

(3) *Nausea Nervosa.*

(Nervous Nausea.)

Nausea is a symptom that, as is well known, is associated with a large number of various diseases. It is, however, also observed as a purely functional disorder, and as such it may take place in such a marked degree and may govern the whole clinical picture that it assumes the character of an idiopathic nervous affection of the stomach.

Nausea nervosa is principally met with in women, in rare cases I observed it also in men. In the former it is almost always associated with general anæmia and chlorosis. It is very liable to develop as a result of menstrual disorders, in the period of puberty, and in some cases in the period of menopause.

Nausea nervosa may be persistent or temporary; it may come and go on one and the same day, and—this speaks particularly for the nervous character of the affection—is independent from quality and quantity of food, indeed from ingestion of food at all. Very frequently, for instance, it occurs that patients of this kind awake with nausea early in the morning.

Psychic excitements may cause the affection, as I frequently observed, or may develop a new attack if the affection is intermittent. *Vice versa,* long-extended bodily rest, diversions, change of air, and travel frequently exert a favourable influence.

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[11] Dujardin-Beaumetz, Traitement des maladies de l'estomac, 1891, P. 326.

On account of nausea the inclination for eating is, as a rule, considerably diminished. In addition, many patients, presuming that their nausea originates in the stomach, condemn themselves to a strict diet poor in proteids and fats, and in consequence fall into a condition of grave subnutrition, without ameliorating their complaints.

In very pronounced cases, nausea may be associated with occasional vomiting.

A great number of cases taught me that nervous nausea may manifest a great variety of degrees; sometimes it may be just hinted at, appearing once in a while, disappearing quickly; in other cases, however, it may occur as an exceedingly stubborn evil, lasting for weeks and months, defying all treatment, whereby patience and medical art are put to a severe test.

Nausea nervosa is very probably due to a condition of changing blood circulation in the brain, which is easily understood in anæmic and easily irritable girls suffering from dysmenorrhœa, and in women in their menopause. That the stomach itself, at least as to all its discernible functions, is intact, is manifested by the fact that in all cases of nervous nausea in which I examined the gastric contents, it showed entirely normal *secretory* and motor function, apart perhaps from the frequently existing superacidity in anæmic patients.

Diagnosis and Differential Diagnosis.

The diagnosis of nervous nausea is usually not difficult. We must, however, not forget that sometimes certain internal causes, such as tapeworms or other entozoa, incipient tuberculosis, reflux of bile into the stomach, pregnancy, diseases of the genitals, etc., may be present. A careful examination is, therefore, indispensable, before we should decide upon the diagnosis of nausea nervosa.

In cases of men, in whom the affection is relatively rare, it is advisable to think first not of functional disturbances, but of central affections (cerebral tumours, pachymeningitis), arteriosclerosis, as well as of diseases of the kidneys (incipient uræmic conditions), and, above all, of diseases of the prostate gland and the bladder. We need not mention that also organic gastric affections themselves (chronic gastritis, beginning carcinoma, atony, ectasia) may sometimes cause symptoms resembling nervous nausea. Only after exclusion of all these affections, the diagnosis of nervous nausea should be approached.

Treatment.

Treatment of nausea nervosa must take its starting point from the existing ætiology to which the affection in the individual case is due. If we are dealing with a purely idiopathic nausea, we will, above all, have to begin therapeutically with the nutrition, which, as mentioned above, is almost always in a reduced state. In many cases I succeeded in obtaining a gradual disappearance of the nausea by amelioration of the nutrition (rest-cure, increased ingestion of food). Suitable hydropathic measures, as well as the constant current, may be added. In all cases, however, I consider separation from the family and the consequent removal of psychical insults, which frequently enough cause or exacerbate the affection, as one of the most indispensable pre-conditions for cure. Such patients are best put in suitable institutions for diseases of the stomach,* or in sanatoria for nervous diseases, which are well governed also in dietetic regards.

The medicinal treatment may sometimes support the physical measures in a valuable manner. Bromides, chloral hydrate in small doses (0.2 to 0.3; three or four times a day), and particularly validol (six or eight drops every two or three hours), have been of at least palliative great service to me. I have, however, not observed any lasting effect, especially in grave cases, from these or any other remedies.

(4) *Gastralgia Nervosa.*

(Cardialgia, Gastrodynia, Pylorospasm.)

By gastralgia nervosa is meant a periodical morbid symptom, characterised by severe pains in the epigastrium and independent from ingestion of food. The pain may be either tolerable, may have a burning, boring, and tearing character, or may be increased to a severe cramp radiating into the hypochondriac regions, to the abdomen and back, and may be associated with the sensation of annihilation and grave collapse, etc. The attack may be accompanied by globus, bulimia, vomiting, dysuria, etc. The paroxysm may last from a few minutes to several hours, may be periodic, in regular or irregular intervals, or entirely atypical without any discernible cause whatever.

Among the causes of gastralgia we must mention: (*a*) causes which concern the stomach itself or its surrounding; (*b*) central causes; (*c*) infections and intoxications; (*d*) reflexes from other organs; (*e*) general neurasthenia and hysteria.

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* [It is remarkable that there are so few special sanatoria for patients suffering from diseases of the stomach and intestines in this country. In the general hospitals, as a rule, not enough attention is paid, nor can be paid, to cases of this kind.]

(*a*) Gastralgias dependent upon the stomach itself and its surroundings may be caused by ulcus ventriculi, some cases of carcinoma, gastritis acida, supersecretio acida, perigastritis, or peritonic adhesions (liver, gall-bladder, spleen, pancreas, transverse colon, and other portions of the intestines, etc.); furthermore, by tumours in neighbouring organs,* I mention particularly the frequent cysts of the pancreas.

(*b*) Among the central causes of gastralgias are, first, tabes [locomotor ataxia]—(crises gastriques), then myelitis (Leyden, Oser), and brain tumours.

(*c*) Infections and intoxications may cause gastralgias; among the former there is particularly malaria, which occurs either as complication of a manifest form or as marked intermittent form. Among intoxications we have to mention nicotine poisoning; in several cases I observed severe periodic gastralgias resulting from it. To them belongs also gout; I observed a very pronounced case of this variety. In such a case the abnormal condition of the blood might probably be the irritant for the occurrence of the attack.

(*d*) Among the reflexes from other organs, the diseases of the genital organs of women deserve the greatest attention. Retroflexion of the uterus, ovarian tumours, affection of the tubes very frequently occasion gastralgic attacks. Of course, it must not be forgotten that idiopathic gastric affection, causing gastralgias, may complicate genital affections.[12] [To this class probably belongs also the close connection between gastralgia and aortic regurgitation.[13]]

Gastralgias may occur, too, in men suffering from genital affections (spermatorrhœa), as Peyer states in his frequently mentioned work.

In cases of splanchnoptosis (especially in ptosis of kidney and liver), gastralgias of more or less marked degrees are very frequent. In a case reported by Renvers[14] severe gastralgic attacks were brought about by intermittent hydronephrosis, probably as a result of direct irritation or traction of the sympathetic plexus.

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* I observed, in a girl of 17 years of age, a tumour which caused severe, almost permanent, gastralgias. On post-mortem examination it proved to be a primary carcinoma of the left lobe of the liver, which was attached to the stomach by firm adhesions.

[12] Compare Panecki, Therapeutische Monatshefte, 1892, P. 79.

[13] DaCosta, Medical Diagnosis, Philadelphia and London, 1901, ninth edition, P. 485.

[14] Renvers, Berliner klinische Wochenschrift, 1888, No. 53.

(*e*) Hysteric and neurasthenic persons often complain of paroxysms of gastralgia without a definite cause being given. The periodic gastralgias in anæmic persons are likewise to be counted among these. As these gastralgias, as I know by experience, are frequently mistaken, they may be briefly discussed.

The paroxysms of pain occur, in the majority of cases, every three or six weeks, more rarely at greater intervals, and last usually two or three weeks, and sometimes longer. The disappearance is sometimes sudden, sometimes slowly remittent. Mostly the patients—almost always women—put the beginning of their trouble at the time of puberty. Occasionally an association with the genital apparatus is evident, as the cardialgias make their appearance either before or after the menstrual period. In other cases patients attribute the onset of the attacks very decidedly to lesser or greater errors in diet, sometimes of a quite definite kind, as, for instance, in one of my cases it was due to the ingestion of candy. In the majority of cases, however, the patients can ingest, without the least trouble, the so-called hard-digestible foods during the interval free from paroxysms, or even in the periods free from pain during the attacks. Appetite also may be good within the time of attack; if the ingestion of food is disturbed, it is more because the general state of health is impaired by the attacks of pain or through fear of causing or exacerbating the pains by ingestion of food. The attacks of pain cannot be the consequence of coprostasis, which, as is well known, is exceedingly frequently observed in anæmia; for in the majority of my cases normal bowel movements have been expressly stated, although during the cardialgic attacks, on account of diminished ingestion of food and increased rest, an interruption of the normal condition occurs.

As to quality, location, and duration of the pains, they are, little deviations excepted, in general alike. The pains begin in the scrobiculus, and remain there pretty well localised; slight radiations toward the left and right hypochondrium occur. The pains frequently occur after ingestion of food, at times following very definitely one or two hours afterward; in some observations pain followed certain dishes, but by no means always the more solid ones. The pain is not influenced by new ingestion, or but slightly so. The pains are designated usually as burning, boring, or oppressing; in some cases—though rarer—as cramp-like. Their intensity is perhaps best characterised by the fact that usually the patients are not compelled to interrupt their occupations; quiet, horizontal position relieves, active motions and exertions exacerbate the troubles.

As to the duration of the single paroxysm of pain, it is likewise pretty constant; it varies between a quarter to half an hour, rarely longer. Sometimes the attacks occur but once a day, but frequently they are repeated several times in the course of a day; the nights seem always to be free of pain. The single paroxysm usually ceases pretty abruptly; rarely it ends in eructations, and only in a single case in my observation vomiting followed; as a rule, the patients feel tired after the attacks and need sleep; the general feeling, however, is very little or not at all impaired. Symptoms of urina spastica I have never observed in my numerous cases. The objective examination of the stomach does not yield any diagnostic points. In all my cases a localised or diffuse area of pain or dorsal algetic points was always absent. Occasionally the right kidney is found to be slightly loose, but never to such a degree that the attacks might be explained as a result of it.

In several cases I made examinations of the gastric contents, expecting to find perhaps chemical or motor abnormalities. Such, however, was not the case. In agreement with the majority of authors, I found sometimes normal, sometimes also increased amounts of hydrochloric acid; in one case there was varying acidity, first pronounced diminution of hydrochloric acid and later normal amount. On account of these varying conditions we are not justified in attributing the appearance of pains to anomalies of hydrochloric acid secretion. Marked deviations from the motor gastric function were not observed.

Besides the above-mentioned gastralgias, there are conditions of true gastrospasm, or rather pylorospasm, due to nervous disorders. To these belong, for instance, pylorospasm of infants, as recently described by M. Pfaundler[15] and Heubner.[16] But in adults also occurrence of true idiopathic pylorospasm cannot be disputed any longer. Schnitzler[17] and C. Brunner[18] have described cases of pylorospasm, which were operated upon, and which must be accepted as purely nervous spasmodic affections.

The case of Schnitzler was that of a markedly hysteric cook, 35 years of age. She stated that various gastric affections had occurred in her family. Patient complained of severe gastric troubles, and stated that repeated hæmatemesis and bloody stools had appeared. On examining the patient, who was considerably emaciated, a hard and slightly movable tumour of about the size of an apple was found repeatedly, not always in the pyloric region. As the patient urged an operation, a laparotomy was made. First, the stomach and pylorus appeared to be sound, then there appeared repeated succeeding peculiar movements, which contracted the pyloric portion and transformed it very accurately to the frequently palpable tumour. The operation had a good effect, lasting for some time; later lighter gastric disturbances reappeared.

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[15] Pfaundler, Wiener klinische Wochenschrift, 1898, No. 45.

[16] Heubner, Verhandlungen des Vereins für innere Medicin, January 21, 1901; Deutsche medicinische Wochenschrift, 1901, Vereinsbeilage, No. 13.

[17] J. Schnitzler, Wiener medicinische Wochenschrift, 1898, No. 51.

[18] C. Brunner, Beiträge zur klin. Chirurgie, 1901, Bd. 29, P. 520.

A clinically exceedingly important cause of gastralgia without any other demonstrable finding is formed by occult herniæ or, according to Lindner,[19] also wide-open inguinal canals. Among the former there are particularly the little femoral herniæ, frequently not larger than a cherry, which cause indefinite pains in the abdomen, colics, flatulency, anorexia, etc., which often enough erroneously sail under the flag of gastric or intestinal catarrhs. The same may also be said of imperfectly developed inguinal herniæ.

[A very instructive case of this kind was observed by me quite recently. A man of athletic build had repeated attacks of pain in the abdomen and of vomiting. Examination of gastric contents showed some increase of hydrochloric acid. After the complaints had lasted for a few months, I found an imperfectly developed inguinal hernia on the left side and a wide-open canal on the right side. I recommended a truss (operation of any kind refused), but patient thought he did not need it. After he had several more attacks of cramps and vomiting a double truss was fitted to him, and he has had no attack since that time, now a period of twenty months.]

The second and equally important group concerns the *herniæ of the abdominal walls*, which occur as lateral and median herniæ. The former rare forms develop almost always in the mammillary line, on either side, and are of various sizes, and may—as Lindner stated—reach the size of a hen's egg. Usually being very much smaller, they are not readily recognised.

Much more frequent are the median abdominal herniæ, which occur in some cases as true herniæ (herniæ lineæ albæ), in other cases as lipomata (properitoneal), and, as Richter already knew a hundred years ago, may become a source of considerable gastro-enteric troubles.

The herniæ occur single and multiple, and are usually located near the xiphoid process or close above the umbilicus. As Kuttner[20] correctly emphasises in an excellent treatise on this type of herniæ, men are afflicted much more frequently than women. I cannot remember having observed any herniæ of the linea alba in women. They also prevail in persons of the labouring class, although I have frequently observed, as Kuttner states, hernia of the linea alba in patients of the well-to-do class. Kuttner mentions, as an ætiological factor, congenital disposition, congenital [or acquired] weakness of the fascia of the linea alba, hereditary predisposition, waste of muscles in marked emaciation, subserous lipomata, and traumatism. According to my experiences, acute traumatism does not play as great a part as continuous mechanical insults [strain], to which the labouring class is particularly exposed, and marked emaciations. I repeatedly observed hernia of the linea alba in cases of cancer of the stomach and œsophagus. The other mentioned causes are decidedly less frequent.

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[19] Lindner, Berliner Klinik, 1892, Heft 49.

[20] Kuttner, Mittheilungen aus den Grenzgebieten der Medizin und der Chirurgie, 1896, Bd. 1, P. 661.

As to the complaints of the patients, it must be stated that they may be entirely absent, or may reach in some cases a slight degree, in other cases an exceedingly high degree. They are distinguished almost always through severe drawing or colicky pains, which are independent from quality or quantity of food, but are greatly dependent upon bodily movements, and still more upon mechanical exertions. The troubles cease very soon if the patient rests in a recumbent posture. Frequently there develops in association with it, as Kuttner emphasised and I also observed, the clinical picture of neurasthenia, hysteria, hypochondria. Very often I observed considerable loss of weight, evidently of artificial kind (due to sitophobia, etc.).

[In this connection I wish to mention a paper read before the twentieth annual meeting of the Association of American Physicians at Washington, May, 1905, which was the last contribution to medical literature by my revered friend and teacher, the late Dr. D. D. Stewart,[21] Philadelphia. This paper was edited by me, and I had helped Dr. Stewart in reviewing German literature on this subject. The paper reports particularly three cases out of his private practice, and I hereby give a synopsis of the cases.

Dr. Stewart's attention was first directed to the subject about ten years ago through a case of his in the Polyclinic Hospital, the first that he had seen, certainly the first he had recognised. The patient, a Russian, was brought to the hospital with a severe attack of abdominal pain and was seen in the out-wards by Dr. T. S. K. Morton and Dr. Stewart. He gave a history of a long-existing, unrelieved gastro-intestinal disorder, the chief symptom of which was recurring colic. Examination of the abdomen disclosed a small hernia in the linea alba above the umbilicus, which was acutely sensitive to touch, and which was evidently the cause of the symptoms.

CASE I.—Case of small supraumbilical hernia of the linea alba, causing recurring, severe gastralgia: E. A. F., slender build, nervous temperament, aged 27, clerk, consulted me July 1, 1904. He had been subject to spells of colic for several years, an attack lasting often four to five days, with intervals of from a few days to several weeks between. Pain had origin in the epigastrium, and thence spread over the whole abdomen. An attack was more apt to occur after eating, especially if he overindulged. The pain might occur without relation to eating, and apparently was very little influenced by the character of the food. Overeating, causing indigestion, would aggravate the frequency and severity of the attacks. There was more or less bowel flatulency; the bowels were quite regular without laxatives; had often been freely purged during an attack, with the idea of decreasing its severity; pain then was rather aggravated than ameliorated. There was no loss of flesh; poor appetite; he got very little fresh air and no out-of-door exercise. Smoked eight to ten cigars daily; moderate alcoholic indulgence.

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[21] D. D. Stewart, American Medicine, July 29, 1905, P. 185-188.

Physical Examination.—Slender-built young man; handles yarns in his business; no blue line, no history of plumbism. Nothing abnormal in chest, save slightly irritable heart (tobacco). Abdomen rather thin, though firm-walled. In the erect posture and in recumbency, there was evident only on careful scrutiny, but easily apparent to the palpating finger, a very small nodule in the linea alba, the size of half an ordinary-sized pea, 1½ inches above the umbilicus. It was firm to the touch, very sensitive to even delicate palpation; it was without impulse on coughing, and irreducible. The stomach was normally placed, liver and spleen outline normal, left kidney not palpable, lower pole right kidney barely so, no hernia elsewhere.

Several examinations made to determine the condition of the stomach, showed mucous subacid gastritis.

Operation was done by Dr. Steinbach, January 27th. The following is the doctor's report as to the findings: "The hernia was occasioned by a somewhat oval opening in the linea alba, about ½ inch in its vertical, and ¼ inch in its transverse diameter. The peritoneum (sac) was adherent to the integument, and a portion of the omentum, in the shape of radiating strands, was adherent to the sac, and therefore, practically to the skin. I excised an elliptic portion of the integument with the adherent peritoneum sac, ligated the adherent portion of the mesentery, cut it off in front of the ligature, and closed the opening by through-and-through sutures, and separate sutures of the linea alba. There was no real strangulation, because the sac could quite properly be said to have had no neck, but the adhering of the mesentery caused it to thicken and form the strands which I mentioned, and this practically gave rise to the pains."

The patient made a good recovery, and at the present writing (May 8th), he reports no attacks since the operation. Flatulency, which had been a marked symptom prior to operation, has now disappeared.

Case II.—M. M., aged 50, merchant; recently seen. Stomach trouble for many years, chiefly nervous in character; gaseous distention of the stomach and bowels constantly present; sensations of fulness and discomfort after meals; bowels regular; appetite good, occasionally "crampy" pains. He is about 10 pounds below his best weight; habits always good; never abused stomach; on the contrary, overcareful. Nervous and hypochondriac. Has been to many physicians in Europe and in this country without relief of symptoms.

Physical Examination.—Chest, nothing abnormal. Abdomen, epigastric hernia sensitive to touch, the size of half a small, flattened shellbark, evident in linea alba about an inch above the navel. Marked gaseous distention of the abdomen. This, he states, has been apparent for years. Stomach slightly dropped; inflation shows inferior border below umbilicus; no dilatation. (Inflation and water tests.) Liver, spleen, and left kidney normally placed; right kidney palpable on deep inspiration, but not abnormally loose. Urine, clear; light amber; acid; specific gravity, 1,008; albumen and glucose, absent. Microscopically, nothing noteworthy.

Examination Stomach-contents.—Cleansing lavage at 9.30 A. M.; 9.45 A. M., 60 grammes bread, and glass of hot water; at 11 A. M., 50 grammes bread, glass of hot water; at 12 noon, stomach empty; 100 cubic centimetres thickish contents; much ropy mucus; total acidity, 55; Günzberg, strong reaction; Toepfer, 30; starch digestion, fair.

The following day cleansing lavage at 8.30 A. M.; 9.45 A. M., ½ pound steak, 2½ ounces bread, 12 ounces weak tea. Three hours after finishing eating, emptied stomach; 200 cubic centimetres well solved; fluid; large amount ropy mucus; amber filtrate; total acidity, 100 (litmus); Günzberg, absent; Toepfer, 53.

Despite the presence of mucus, acid gastritis with atony, and the absence of direct symptoms resulting from the hernia, I regard the latter largely as the cause of the symptoms. The hernia had existed at least sixteen years, about the time of the onset of the ailment. It is soft to the touch, at times a slight gurgle is evident in it, as if it might be a gastroepiplocele; it is irreducible. The patient has been under observation two months; the gastritis received active treatment; morning lavage, with hot soda solution, followed by cool physiologic salt douche; silver nitrate douche, or silver vitellin employed two or three times weekly for a month. Menthol valerianate for the control of nervous belching used without result, as was naphthol and charcoal, to discover if the flatulency was of fermentative origin. Bitter tonics and nervines were administered, all of which are usually helpful in the condition present. Although there was considerable improvement manifest in the gastric condition as concerns diminution in mucus production, and lessening in atony, the flatulency, nervous belching, and general gastric discomfort persisted. Despite urging, the patient refuses operation.

CASE III.—The following case was referred to me by Dr. B. Segal, for an opinion. The doctor stated in his note of reference that the patient "had been suffering from pain and distention of the stomach for a number of weeks. Treatment gives him temporary relief only. I suspect malignant trouble."

A. G., aged 50; attacks of cramp-like pain of great severity in epigastrium; through to the back; present for at least five weeks; then with onset of pains, at the height of their severity, he had spells of vomiting, which now no longer occur, although pains still as pronounced.

Examination of abdomen disclosed a small, fixed, epigastric hernia in the linea alba, a little below the xiphoid cartilage; it has a circumference about that of a silver quarter of a dollar; it is soft, and suggests a gastroepiplocele. Pain has origin in this spot, and then radiates through either hypochondrium to the dorsal region. The hernia is exquisitely sensitive to touch; there are no other gastric symptoms; appetite is good, and bowels regular.

The patient was returned to the doctor, with an opinion that the hernia was undoubtedly the cause of his trouble. The patient refused operation.

In these cases the hernia was small; in the first it was barely apparent to the eye, while in cases two and three it was, though small, fairly evident on inspection, with the patient in the erect posture. It is cases such as these which are the most troublesome, since the apparent triviality of the protrusion, even if it does not escape recognition, often does not suggest itself to the physician as a likely source of the malady.

The occurrence of epigastric hernia, the majority of which is in the linea alba, was originally described by Garengeot,[22] under the name of gastrocele. It furnishes somewhat less than 1 per cent. of all varieties of hernias,[23] and is far more common in the male than the female. It is more usual in adult life than in infants or children, and its occurrence in the majority of cases is believed to be due to a congenital or acquired defect in the fascia[24] of the linea alba, as a result of which trauma and strain produce a slight diastasis of the recti. Into this either a nodule of subperitoneal fat (preperitoneal lipoma united by a pedicle to the peritoneum) makes its way; or more commonly, a portion of the omentum,* which rarely may be accompanied by a portion of the small intestine, and more rarely by stomach. A strain, as by heavy lifting, or some unusual form of exercise, violent vomiting, or coughing, producing a sudden rise in intra-abdominal pressure, is the probable exciting cause. These forms of hernia, of which one is the rule, and of which two are unusual, whether preperitoneal fat or omental, through their nerve connections and especially through their attachments to the subjacent structures, and to the stomach** or liver, and their fixation by adhesions about the orifice of the ring and its under surface, rarely fail to originate early symptoms after their appearance. There may be no apparent fixation of the hernia at the ring, a portion of omentum being intermittently incarcerated, and producing recurring pains only through its presence. With relaxation of the recti in recumbency the hernia may disappear, as in a case recently observed: The small separation in the linea alba, or immediately to one side, apparent on careful palpation, then furnishes the clew to its existence. The symptoms are commonly as described in the foregoing cases and in those to be related, and are usually referred to the stomach, whence a diagnosis of some form of gastric disorder, gastritis, gastralgia, ulcer, or even gastric carcinoma, is made by the unwary, or the case may be looked upon as one of gall-stones.

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[22] Memoires de l'Academie Royale de chirurgie; memoire sur plusieurs hernies singulieres, 1743.

I recently saw a case through the kindness of Dr. Deaver, in which the symptoms—severe recurring attacks of gastralgia and enteralgia, with vomiting and debility, and neurasthenia of high grade—had been previously attributed to gall-stones by a well-known surgeon, and the diagnosis was concurred in by a clinician of wide experience. On this account, in the operation for the removal of the epigastric hernia by Dr. Deaver, the gall-bladder was also explored, but found entirely free from disease.

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* A comprehensive account of the pathologic anatomy of epigastric hernia has been given by H. A. Lathrop (Boston Medical and Surgical Journal, February 25 and March 4, 1897). Lathrop points out that as in the epigastric area there are no preformed openings, the hernia must make its way through a rent in the tissues of the linea alba, and that a common point of origin is that between the umbilicus and the xiphoid cartilage at which the round ligament and paraumbilical veins leave the abdominal wall, on their passage to the liver.

** It is especially the pull or drag of the omentum on the stomach in these cases which tends to originate the gastric symptoms.

[23] Berger, Traite de Chirurgie, 1895, noted 137 cases in 16,800; and Bohland, in Policlinic in Bonn, 33 cases in 3,420; Kuttner.

[24] Warren Gould, International Text-book of Surgery, 1902, P. 510.

In this case there was slight reason for confusion, the hernia was large, and although it had existed for a long time without special direct symptoms, the recurring and persistent attacks of severe colic following immediately after exercise involving unusual physical strain, indicated the source of the pain. The patient was of neurotic temperament, and had become the subject of hysteroidal attacks while suffering from nervous prostration, the result of mental overapplication and family worry. Later, when in fairly good physical condition, an attack of intense abdominal pain immediately followed unusual muscular strain, as above stated, and these attacks had continued (and entirely replaced the former hysteroidal seizures) with increasing frequency for eight years. Morphine was required for their relief. The character of none of the seizures suggested gall-stone disease; they were distinctly gastralgic and enteralgic, and if not a result of the hernia, could only be regarded as of neurotic source. An interesting feature was the persistence of the attacks of abdominal pain for some months after operation, and then their gradual cessation; their long existence before removal of the hernia having, in a neurotic subject, established a pain habit not easily broken. The case in this respect seemed analogous to those of Jacksonian epilepsy, in which a too tardy, though thorough removal of a coarse brain lesion does not readily cure the firmly-established fit habit. The protrusion was still evident for some months after operation, but now has not been apparent for a long while, although its former site is still extremely sensitive to slight pressure. The attacks of pain diminished in severity following operation, but did not finally cease for some time. None has now occurred for over five months.

Lathrop[25] related an instructive case in which attacks of jaundice occurred complicating abdominal pain, the result of an epigastric hernia, and caused the diagnosis of gall-stones to be made by several physicians.

The patient, when a rugged boy of 18, fell upon a sharp-pointed stick, injuring the epigastrium. Collapse followed, and for some weeks local pain. Six years later sharp pains in the epigastrium, with other gastric symptoms, especially after eating, appeared and continued with varying severity. For ten years from this time he suffered from moderate gastric symptoms, accompanied by pain and tenderness in the epigastrium. Twenty years before Dr. Lathrop's report, the attacks of pain and vomiting became more frequent and severe, and then a small tender swelling was noticed above the umbilicus. This tumour increased until it became the size of a fist, but was commonly reducible with coincident alleviation in symptoms. Five years later, in addition to the attacks of pain and gastric symptoms, coincident with the appearance of the hernia, he began to have chills, elevation of temperature, and a marked degree of jaundice. The attacks were without periodicity, from a week's interval to occasionally three months, provided the hernia was retained in position, although persistent distress after eating and a dull ache and occasionally sharp pains in the epigastrium were noted. Inspection with the patient lying on the back showed nothing abnormal, except a little fulness of the skin in the median line, half way between the xiphoid and the umbilicus, which became prominent on coughing, and when fully protruded formed a mass the size of a hen's egg. The mass could be reduced, except a small portion which was adherent to the ring. The ring admitted only the tip of index finger. Operation showed the omentum firmly adherent around the whole inner circumference of the ring, and continued inward toward the pyloric end of the stomach (greater omentum), and thence to the liver (lesser omentum) as a fairly firm band. On severing this from the ring it was found to contain the round ligament of the liver (obliterated umbilicus vein), and a portion of the falciform ligament and the great omentum.

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[25] *Loc. cit.*

Seven months after operation the patient was reported "entirely relieved of all his old symptoms, and only regrets that he was not operated on twenty years before."

Lathrop notes that jaundice has been recorded six times as a complication in epigastric hernia in cases in which at operation the gall-bladder and ducts were normal. The patients were operated on for presumed gall-stones. "In all the cases there was an adherent omental hernia, the tension of which may have obstructed the gall-bladder at times."

Bergmann,[26] of Berlin, in a clinical lecture in 1894, stated that he had encountered more than a dozen cases of epigastric hernia "in which the best specialists of Berlin and Paris had diagnosed gastritis, gastric ulcer, and cancer, and had previously employed lavage, rigid diet, and other approved modes of treatment, without result." Hernia in the linea alba had later been discovered, corrected, with a cure in each instance; and König,[27] in his text-book on Surgery, relates similar instances.

In this particular, Davis reports a very interesting case, which for a number of years he and other physicians had more or less under observation, and in which he, throughout this period, without thorough examination, had treated for gastritis, presumably the result of alcoholism. The man had recurring attacks of very "intense gastric pain, vomiting, great nervousness, and cardiac depression. The ingestion of but a teaspoonful of water would aggravate his suffering and provoke vomiting. Morphine, hypodermically, alone gave relief. The seizures usually followed close upon the heels of debauches, lasted from three days to a week, when the symptoms disappeared as if by magic." But it was later noted that many of the seizures were dissociated with alcohol, and as he was rheumatic, it was presumed by another attendant, and in a measure then coincided with by Dr. Davis, that the affection might have origin in "a rheumatism of the muscular fibres of the stomach." Some eight years after he had first been seen by Dr. Davis, but when he had not been for some time under the latter's observation, an abdominal examination by Dr. Davis disclosed a small hernia of the linea alba "not larger than a butter bean," about 1½ inches below the xiphoid appendage. Of the existence of this, the patient had been aware for years, and had been told by previous attendants whose attention had been directed to it, that it was a small cyst and of no importance. Operation by Dr. Davis showed an adherent omentum continued in "the form of a band to a greater curvature of the stomach not far from the pylorus." There was no recurrence of the attacks, the patient making a complete recovery.

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[26] Quoted by Davis, Annals of Surgery, i, 1895.

[27] Quoted by Davis, *loc. cit.*

Aaron[28] relates a case in which the symptoms of presumed gastric catarrh had existed for four years. There were, with intervals of freedom from symptoms, recurring attacks of nausea and vomiting, and epigastric pain; anorexia and a loss of 28 pounds in four months due to the peristent inability to retain food. The removal of an epigastric hernia, about the previous existence of which the patient was unaware, brought about a speedy recovery, with no return of symptoms in the lapse of five years.

W. H. Bennett,[29] in a clinical lecture, entitled "Deceptive Abdominal Pain in Latent Hernia, Especially with Reference to the Surgical Aspect of Certain Cases of Indigestion," recites two very interesting cases of long-existing and improperly treated "dyspepsia," due to unrecognised epigastric hernia in the linea alba, and cured by their removal.

One, a woman, aged 32, had "for ten years been constantly a victim of very acute dyspepsia," as she described it. Cramp-like pain after food, especially that which was indigestible, was the most prominent symptom. She had consulted a variety of physicians, and by all had been treated for dyspepsia. Bennett, at the termination of an examination of the abdomen, and then somewhat accidentally, discovered in the linea alba, about half way between the ensiform appendix and the umbilicus, a small, rounded, movable nodule, about the previous existence of which she or her former attendants had no knowledge. From this the pain had origin and radiated. Dr. Bennett remarks as to the somewhat accidental discovery of the hernia, that he examined her as carefully as he could, and was bound to say he could at first find nothing to account for the symptoms. Finally, however, as he happened to be palpating the middle part of the abdomen, the patient suddenly exclaimed, "that is the spot at which the pain begins." The removal of the offending omental protrusion, which was of no greater size than a large pea, completely cured the long-existing indigestion.

The second case reported by Bennett is also well worth reciting:—

It is that of a man in whom there was discovered about an inch above the umbilicus a small, rounded, movable nodule, the size of a cherry. The patient had suffered for a long time from "intense rolling and cramp-like pains about the middle of the abdomen, which always followed the taking of food." He had been treated for dyspepsia, and all sorts of drugs had been prescribed without relief of symptoms. Operation disclosed a rounded nodule of fat (a congenital lipoma) which, terminating in a thin stalk, was continuous with the subperitoneal fat and passed through a small opening in the abdominal aponeuroses not larger than a No. 8 English catheter. The special interest in the case is in the fact, as narrated by Bennett, that it demonstrated conclusively that pains and discomfort indistinguishable from those produced by an omental hernia in this situation may be caused by protrusion of a mass of extraperitoneal fat.

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[28] Medical Record, 1897, 52.

[29] The Lancet, September 15, 1894.

Lindner,[30] in a monograph on abdominal hernia, relates cases similar to the foregoing of previously unrecognised epigastric hernia in the linea alba.

In one, an army officer, the symptoms had existed for seven years, the hernia having probably been originated from a strain during violent exercise. His attendant then noted resistance in the epigastrium, and an ointment was prescribed. The patient continued to have violent epigastric pain, in one attack of which he fainted; in another there was considerable rise of temperature. He was often incapacitated, and consulted many physicians, all of whom diagnosed gastric ulcer. The symptoms continuing, he went from one physician to another; was put on various diets and bath cures, and had frequent lavage; for a year before consulting Lindner he had been given daily massage of the epigastrium under the most violent pain, with an idea to its amelioration. He grew progressively worse, and at the expiration of seven years after the onset of symptoms, he consulted Lindner, who found a small hernia in the linea alba, located "a hand's breadth above the umbilicus." Operation resulted in absolute cure.

Kuttner, in Berlin, in an instructive paper[31] on "Digestive Disturbances Caused by Various Forms of Hernia, Especially Those of the Linea Alba," refers to the cases of Lindner and others, and cites the following very interesting instance of previously unrecognised small supraumbilical hernia, which had been productive of various ill symptoms:—

A merchant, aged 56, for two years had had paroxysmal attacks of colic. Pain radiated from the epigastrium to the back, girdle-like; commonly one to two hours after food, especially after solid food; colicky pain on exertion, and especially on bending or lifting, ameliorated on assuming the recumbent posture. Lately gastric pain had been constant; no vomiting; bowels sluggish; tense abdomen, due to meteorism. Headache, vertigo, hypochondriasis were present. No tumour was ever noted by his physicians. Various forms of diet were tried. He was sent to a stomach specialist; normal gastric chemism found. Diagnosis: nervous indigestion. Treatment accordingly; no benefit. As he had had lues twenty years before, inunction cure for syphilis was tried unavailingly. He was finally seen by Kuttner. A small hernia the size of a lentil was discovered in the linea alba, two fingers' breadth above the umbilicus, irreducible and very sensitive to touch. Operation resulted in restoration to health.

Kuttner remarks that these cases might readily be mistaken for various abdominal disorders, and especially for simple gastralgia and enteralgia, for gastric ulcer and carcinoma, for cholelithiasis, and for gastric neurasthenia and hypochondriasis.

Although I have long had in mind the importance of this subject, I was, until I began this report, unaware of the extent of the literature on epigastric hernia, and held with Davis, that its dearth was ample reason for non-recognition of these cases showing direct and reflected gastro-intestinal symptoms. Davis remarks that "the leading American text-books on surgery do not mention it at all, or dismiss it with a line. Careful search through a large number of American surgical works has failed to discover a word to indicate the severe gastric symptoms which a slight hernia in the linea alba may cause." This fact probably accounts for the paucity of knowledge on the subject shown by surgeons* and clinicians in this country. Abroad it has somewhat received the attention it merits, but not at all generally, either by clinicians or surgeons. Doubtless, nowhere is it commonly recognised that even a very small hernia in the linea alba may be the source of obscure and distressing abdominal symptoms, nor is the importance of a search for these small hernias as a routine procedure in the examination of the patient very generally in the mind of the clinician.

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[30] Berliner Klinik, 1892, Heft 49.

[31] Mittheilungen aus den Grenzgebieten der Medicin und der Chirurgie, Erster Band, 1896.

A quite complete bibliography to 1897 is given in the paper by Kuttner, and in that of Lathrop. Since these, a few other reports of cases have appeared, which are chronicled in the "Index Medicus."

It is a strange coincidence that the above-mentioned four cases of epigastric linea alba hernia occurred in persons of the Jewish race. Whether it was a mere coincidence, or whether there is any special ætiological factor, I am not prepared to say. Dr. Segal's patient was a carpenter, and a very hard worker. Dr. Boas's remarks as to the occurrence of this variety of hernia in persons of the well-to-do class refer also to Stewart's case of the young man aged 27 years.]

Diagnosis and Differential Diagnosis.

As we defined above gastralgia not as a disease, but as a symptom of a disease, we are obliged, strictly speaking, to determine in each case the real basis for the gastralgia. According to experience, a number of diseases are known to us in which gastralgias form a more or less prominent part, such as, for instance, in the above-mentioned herniæ and in anæmia. In other and not infrequent cases the fundamental affection remains either obscure or is not found at all; we must, in consequence, content ourselves with the symptoms. Sometimes, however, the determination whether a gastralgia or an entirely different disease is present, becomes difficult.

The gastralgic attack in itself has nothing typical when compared to many other paroxysms of pain in the abdominal cavity; the manifestation said to be characteristic by various writers, that the pain is reduced by pressure, is just as deceptive as the statement that the attack experiences relief from the galvanic current (anode at the point of pressure, cathode at the vertebral column). By exclusion we will usually arrive at the right diagnosis. Only if we succeed in palpating the pylorus alternately in its relaxed and contracted state, diagnosis of pylorospasm becomes certain. The assumption of a nervous spasm is only plausible when organic affections are out of the question, something that can be proven but seldom. As to differential diagnosis, there are concerned intestinal colics, cholelithiasis, ulcus ventriculi, intercostal neuralgia, and renal colics.

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* Warren and Gould (International Text-book of Surgery, 1892) state that "far too little prominence is given to this variety of hernia in the text-books on surgery."

Intestinal colics are differentiated from gastralgias by the fact that they are associated with extensive tympany of the abdomen, and are preceded by temporary or persistent irregularities of the bowel movements. Abundant passage of flatus or abundant defæcation is immediately followed by alleviation of the troubles.

In typical cases the differentiation of *gastralgia* from *cholelithiasis* is easy; in the latter there is enlargement of the liver, which is exceedingly tender on pressure; a *tenderness* is usually present also at the *posterior area of the liver*, about in the vicinity of the twelfth thoracic vertebra, in some cases very close to it, in other cases a few centimetres distant; if icterus is present, there can hardly be any doubt. In less typic cases the differential diagnosis may often become very difficult. Of importance is also the fact, which may be elicited in anamnesis, that in cases of gallstone colics there occur dyspeptic symptoms of various degree (particularly often stubborn constipation), and that a descernible connection manifests itself between the attacks and errors in diet. Gastralgias may also be mistaken for so-called liver neuraglias; if the latter are essentially confined to the hepatic area, a differentiation is possible, otherwise it may be exceedingly difficult.

The differential diagnosis between *gastralgias* and *ulcus ventriculi* is not difficult in pronounced cases: the circumscribed algetic point, epigastric as well as dorsal, the independence of the attacks of pain from the quantity and quality of the foods, the alleviation of pain while resting and exacerbation while moving about, the stationary nature of the attacks of pain, etc., speak in favour of ulcer. In atypical cases (see page 434) a decision may become impossible or not be more than a probable diagnosis. I emphasise here the already-mentioned (page 432) diagnostic significance of an algetic point which is situated in the region of the tenth to twelfth dorsal vertebra, close at the left of the vertebra. If any ætiologic foundation for gastralgias (see below) is absent, and the pains are stationary, we should, according to v. Leube's advice, though other symptoms are missing, think of an ulcer, and treat the disease as such. Although the good effect of a regular ulcer treatment does not decide a diagnosis absolutely, it certainly supports it to a high degree.

In some cases *intercostal neuralgias* may cause symptoms simulating gastralgia. A careful observation of the painful area concerned, either by the patient or the physician, furthermore the permanent tenderness along the entire course of the intercostal nerves, or at least of a few points, is usually decisive.

Renal colics may likewise occasionally simulate gastralgia; particularly as the patients are frequently not capable after the attacks of making known the exact painful area. Through accurate examination of the urine, and the search for blood or concretions in the urine after the attack, the diagnosis will be facilitated.

The above-mentioned frequent occurrence of occult herniæ, as well as of herniæ in the abdominal walls, obliges the diagnostician in all cases of gastralgias with obscure ætiology, to palpate carefully all hernial portals as well as to pay attention to possible herniæ or lipomata of the linea alba; they are easily recognised by the experienced practitioner. We must, however, carefully consider whether, in the individual cases, they are just a coincidence or whether they represent the real cause of the affection.

Treatment.

The treatment of the gastralgias requires, above all, the consideration of the primary cause. This is possible in a large number of cases (malaria, abuse of tobacco, genital affections, gout, enteroptosis, hernia, retroflexio uteri, anæmia, and others); in other cases we must content ourselves with the treatment of the gastralgic attacks themselves. One of the best-known and most valued measures is given in the application of hot compresses or heat in the shape of hot water bags, thermophores, etc. Perhaps in a few cases the constant current (anode at the epigastrium, cathode at the vertebral column or in the left axillary line, from five to ten minutes) may be tried as a curative measure, although the effect is usually not very great, quite aside from the fact that an electric apparatus is not always at hand. Oser[32] occasionally also observed that the pain ceased on application of the faradic current. In cases of severe kind the subcutaneous injection of morphine (0.02) or codeine (0.04) is the best remedy; or suppositories of morphine, opium, or belladonna may be recommended after the following formula:—

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[32] Oser, Die Neurosen des Magens und ihre Behandlung, Wien and Leipzig, 1885.

℞ Extracti opii	0.05
Extracti belladonnæ	0.03
Butyri cacaonis	q. s.

u. f. suppositoria No. x.
Sig.: One or two suppositories during the attack.

In some, particularly lighter cases, antipyrine in doses of 0.5 to 1.0, alone or combined with small doses of a narcotic, has proven very useful. Oser (*l.c.*) recommends atropine (0.0005) twice or thrice a day. In quite light cases good results may be had from the administration of sulphuric ether (10 to 25 drops), or ethereal tincture of valerian (15 to 20 drops), or chloroform water (1:100) by the tablespoonful.

In a case of severe gastralgia, Malbranc,[33] on Kussmaul's advice, applied the gastric douche with warm carbonated water with excellent effect, but the method does not seem to have found many followers. We must not underrate the application of hot poultices or mustard plaster, or, finally, the local anæsthetic (chloroform, ethyl chloride, cocaine, etc.).

In gastralgia, due to anæmia, I observed very frequently excellent results through amelioration of nutrition by a rest-cure combined with food rich in proteids and fats. Likewise I saw favourable results from living (during three to six months) in the country or mountains. As to the treatment with iron preparations, I am rather sceptic.

In cases of gastralgia, due to herniæ, one may first make a trial with a well-fitting truss; but where a good result is not obtained, surgical treatment is advisable. Particularly in cases of linea alba herniæ I frequently observed numerous and permanent cures from surgical intervention.

(5) Painful Emptiness of the Stomach.

(Gastralgokenosis.)

By "painful emptiness of the stomach" (gastralgokenosis, derived from γαστραλγεια, gastric pain, and κενός, empty) I understand, as the name says, the occurrence of pains in the stomach or painful pressure in the stomach region, which appears exclusively when the stomach becomes empty and which is relieved by ingestion of food. The affection may be periodical or permanent. According to my view, we have to deal with abnormally severe contractions of the pylorus, which become severer towards the end of digestion after liquefication of the ingesta, and probably become retarded after new introduction of food. I had the opportunity of observing this neurosis, which has never before been described, very pronounced in several cases; a slight degree of the same seems to occur more frequently, quasi as a transition from normal to abnormal conditions of irritations. [Hemmeter[34] states he has never seen a case that corresponds to Boas's description of this malady, and suggests that it is probably a gastric hyperæsthesia associated with hyperperistalsis and a strong secretion of HCl. I believe there are cases which can be classified under the term of gastralgokenosis, cases in which a special high degree of hyperperistalsis has not been found, and in which the hydrochloric acid reaction was not above the normal amount; I have particularly one case of a woman in mind, in which the primary cause perhaps might have been an occasional or rather periodical abuse of alcohol.]

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[33] Malbranc, Berliner klinische Wochenschrift, 1878, No. 41.

Diagnosis.

The diagnosis is made from the symptom-complex, which was manifested, according to my experience, as follows: one to two hours after meals an exceedingly painful pressure in the epigastrium makes its appearance, and may also radiate toward the back. Under some circumstances the pain may be so severe as to embarrass the respiration of the patient. The pain appears during the day and also at night. The paroxysm usually is of short duration, a quarter to half an hour. This pain is *never associated with the sensation of bulimia*, although it is relieved, apparently in a very short time, by the ingestion of milk, cake, or other substances. The quality of the food previously ingested is not without influence in some cases. In one of my patients an attack developed every time he drank white wine or champagne, or ate cake; in others, a similar influence was not observed. Some of my patients state that the affection becomes severer after excitement, anger, irregular mode of living. According to my observations, the malady may be stationary or periodical; there are, moreover, frequently more or less marked signs of general neurasthenia, though never to a particularly high degree.

The examination yields, against all expectations, by no means excessive amounts of hydrochloric acid; on the contrary, the chemical as well as motor functions are found to be perfectly normal. In two of my cases there was, besides the "painful emptiness of the stomach," a stubborn constipation; after the latter was relieved the former disappeared.

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[34] Hemmeter, Diseases of the Stomach, Philadelphia, 1902, third edition, P. 803.

Differential Diagnosis.

The affection, on superficial observation, could be mistaken for bulimia, but the fact that ravenous hunger is not even hinted at, and much less appears as a characteristic symptom, points to the difference of the two conditions. The so-called superacidity also could be assumed as the basis for the malady, but, as mentioned above, a real superacidity was not found in my examinations. Though periodic occurrence of the affection and the perfect well-being in the intervals speak against simple superacidity, we must consider whether gastralgokenosis may not be caused by superacidity, which may be only temporary. According to my experience there are certainly cases in which, in spite of the most careful examination, deviations in the functional condition of the stomach cannot be found.

The following two observations may illustrate and complete the clinical picture:—

Case I.—Banker, no hereditary history, besides children's diseases was never ill, with exception of sciatica. The present malady has lasted for ten years, and made its appearance first at long intervals, now at shorter intervals. The attack begins with nausea, salivation, and yawning, followed, one or one and a half hours after a small meal, or three hours after a larger meal, by painful pressure in the epigastrium. The pain is sometimes so severe as to embarrass the respiration of the patient. The attack is not associated with feeling of hunger. The pain is relieved in a short time by the ingestion of cake [biscuit, cracker] or wine, milk, etc. Otherwise appetite is good; bowels regular; weight of body constant at 100 kilos. Status præsens: strong, healthy-looking man, with normal circulatory and respiratory organs; no albumen or sugar in urine; epigastrium not tender; borders of the stomach normal, no splash-sound; liver edge two centimetres below costal arch; region of gall-bladder not tender. Examination of gastric contents one hour after test-breakfast yields normal amount of food-remnants; decided HCl reaction; total hydrochloric acid = 0.21 per mille.

Case II.—School-teacher, descended from healthy parents; always healthy in childhood (no children's disease), and of strong constitution. For the last year there exist peculiar gastric troubles, which occur periodically every six or eight weeks, and then disappear for several months, only to recur without any apparent cause. The troubles make their appearance usually during daytime, more rarely during the night, and always when the stomach is empty; they are relieved by ingestion of a small quantity of milk or bread, but not at all or only slightly through soda. In the beginning of the malady, the attacks of pain were accompanied by vomiting, which, however, did not occur during the later paroxysms. Feeling of hunger or heartburn is not associated with the attacks. I mention especially from the status præsens: well-developed, strong man, no neurasthenic symptoms; patellar reflexes slightly exaggerated; slight tenderness in epigastrium, no atony. Fasting stomach: a few (two or three) cubic centimetres of greenish muriatic contents. Test-breakfast one hour after ingestion; normal amount, well chymified; decided HCl reaction, total acidity = 56.

Treatment.

In order to avoid the occurrence of the attacks it is advisable, as the patients themselves usually have already experienced, not to allow the stomach to become entirely empty. The patients had better always carry with them a few cakes [crackers] or a little bottle of claret or milk. [I always recommend for this purpose a small bottle of milk lunch tablets or v. Mering's vigor chocolate.] Small doses of opium extract (0.005 to 0.008), it seems to me, have frequently been of more than temporary service for allaying the attacks; small doses of bromides also have been of use. [Bromides, I believe, may be useful in cases of neurasthenic character, but usually when there is even a semblance of hyperchlorhydria I would advise against the use of bromides by the mouth, because in many cases bromides will rather exacerbate than diminish the condition; the bromides are better given by the rectum.] Possible other troubles (constipation, neurasthenia) must, of course, not be neglected.

(6) Stomach-burn.

Besides heartburn, there occurs, according to my experience, a neurosis, not heretofore described, which may be designated as "stomach-burn," according to its essential symptom.

"Stomach-burn" is located, in distinction from heartburn, not in the cardiac region, but at the fundus of the stomach; but it may extend along the œsophagus to the throat.

As in all other neuroses, stomach-burn may occur as a permanent or periodic affection, but the periods themselves—and this is of characteristic importance for the conception of the trouble—are entirely irregular and incalculable. Though the mode of living may be the same, they may occur every day, and then disappear abruptly.

Excitement provokes the stomach-burn; rest and diversion may cause its disappearance. Stomach-burn is absolutely independent from the ingestion of food, and from quality and quantity of the ingesta. Even on an empty stomach or on light diet, the painful sensation may exist. Astonishing also is the inefficacy of alkalies, and especially of sodium bicarbonate.

Diagnosis.

The diagnosis is given by the clinical picture just described; moreover, it must be ascertained through examination of the stomach-contents that superacidity is not present. The finding of other neurasthenic signs will essentially support the suspicion of a gastric neurosis.

In order to illustrate the clinical picture of stomach-burn I shall report the histories of two patients, as follows:—

Case I.—Miss M. K., 48 years, teacher. Patient has been complaining for twelve to fourteen years, and at intervals of some weeks or months, of a sensation of "burn" as if fire were in the stomach. The burn radiates to the arms, shoulders, and legs. In the intervals the patient's condition is perfectly good, and she can eat with impunity any kind of food except acid food. Since January, 1900, permanent burning, day and night, has made its appearance as result of excitement, and independently from eating. The same decreases when patient is moving about, increases when at rest. Appetite continues to be good, bowels are regular. Loss of weight during the last two months, 2 kilogrammes. On examination, perfectly normal conditions of circulatory and respiratory apparatus are found. The abdominal examination yields general enteroptosis, displacement of the right kidney (second degree). Stomach-contents normal as to motor and secretory function.

Case II.—Mr. Str., 28 years of age, merchant in Hamburg; family history good; patient was never ill. For the previous nine years he has had gastric troubles, due, as he states, to frequent drinking of ice-water while in America. The troubles were manifested in the beginning by pains in the stomach. These pains formerly occurred particularly in the morning before partaking of food, but disappeared after breakfast. Later, pain occurred, according to his statement, especially after eating cake or pie, and after food difficult to digest. For the last three months pain in the stomach has not been present, but there exists *burning in the stomach, which often extends along the route of the œsophagus upwards to the throat.* The burning is independent from meals; administration of sodium bicarbonate sometimes brings relief, at other times it does not. Patient lost about 10 pounds in weight. Appetite good, bowels regular. Status præsens: well-developed man in defective state of nutrition; skin and mucus membranes pale. Prompt reaction of pupils to light and accommodation. Reflexes of tendons active. Examination of internal organs shows normal conditions. Colospasm of the sigmoid flexure. Urine without albumen or sugar.

February 19, 1901.—Test-breakfast contains mucus, free HCl 7; total acidity 30. Mathieu-Rémond 150 cubic centimetres.

March 1, 1901.—Test-breakfast anacid.

March 8, 1901.—Test-breakfast anacid.

March 15, 1901.—Test-breakfast: free HCl 8; total acidity 30.

Patient complained, during his stay at the clinic, in the first *two weeks* daily of burning in the stomach, occasionally also of "heartburn"; later the "burn" occurred less often. It was independent from the meals, and was not influenced by drinking of Bilin water, or eating of cake (crackers). Patient gained 5.5 kilos within twenty-four days.

Treatment.

The treatment has the task of strengthening the whole nervous system, of improving the state of nutrition, and of removing the stomach-burn. As to the latter, hot poultices and bromides (0.5 three times daily) have been of service. Chloral hydrate also, in small doses, is worth a trial.

[Cases of this kind are sometimes very stubborn to treatment, as the complaint is frequently of a decided neuropathic nature; general curative measures and suggestion may be of great value. In one case the best relief from the "burn" was afforded by the ingestion of ice cream.]

II. Motor Neuroses of the Stomach.

(1) *Rumination or Merycism.*[35]

Preliminary Remarks.—Rumination is either an idiopathic affection or an accompanying symptom of a disease. The malady is characterised by the fact that sooner or later after partaking of food a part of it returns into the oral cavity of the patient. According to its taste it is either reswallowed or spit out. The whole act passes off without any exertion whatever, without disgust or any feeling of nausea. This rechewing, from which the process derives its name, is not necessarily associated with it; indeed, as a rule, it is found only in the insane and idiotic. Körner[36] distinguishes a form of rumination with dyspepsia, and another one without dyspepsia. We are hardly mistaken in the view that the dyspeptic disturbances are usually of secondary nature. This corresponds to the fact that the examination of the gastric contents in patients afflicted with this affection shows results of most varied kinds (Alt,[37] Boas,[38] Jürgensen,[39]

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[35] Compare as to its historically interesting literature, the following treatises: Peyer, Merycologia sine de ruminantibus et ruminatione commentarii, Basileæ, 1685; Schneider, Das Wiederkäuen beim Menschen, Heidelberg Medicin. Annalen, 1846; Pönsgen, Die motorischen Verrichtungen des menschlichen Magens, Strassburg, 1882, P. 124 *et seq.;* Bourneville et Séglas, Du mérycisme, Arch. d. neur., 1883; Johannessen, Ueber das Wiederkäuen beim Menschen, Zeitschrift für klinische Medicin, Bd. 10, P. 274.

[36] Körner, Deutsches Archiv für klinische Medicin, 1883, Bd. 33, P. 544.

[37] Alt, Berliner klinische Wochenschrift, 1888, No. 26.

[38] Boas, *ibid.*, No. 31.

[39] Ch. Jürgensen, *ibid.*, No. 46.

Einhorn,[40] Leva,[41] Freyhan,[42] Decker,[43] G. Singer,[44] Mayet,[45] Linossier and Lemoine,[46] [D. Riesman[47]], and others). Einhorn and Decker found perfectly normal amounts of hydrochloric acid and normal motor function. Among my numerous cases of rumination I could not find any case of pronounced ectasia, such as has been reported by others. From rumination developed to the highest degree (which, as a case reported by Oser[48] teaches, may under some circumstances lead to the gravest disturbances of nutrition) down to occasional and temporary rumination, there are intermediate stages of all varieties.

According to my opinion, however, it is not yet certain whether rumination may not occur as a sequela to organic gastric affections; at least I observed a few patients afflicted with rumination, who were not especially troubled by the rumination itself, but who told of other disagreeable disturbances (pressure, fulness, pain, nausea). Whether these sensations are not primary, and the rumination must rather be conceived as secondary, still needs explanation.

[D. Riesman reports two very interesting cases in which hydrochloric acid was absent. In one case only a trace of lactic acid was found, in the other case at one time much lactic acid; these examinations, however, were not made after a regular test-meal. In the latter case there was a distinct three-layer separation in the gastric contents, although the report reads that a dilatation of the stomach was not present.]

The views about the ætiology of rumination vary. Some investigators (Dumur, Lebert, Oser, and others) assume a permanent paresis of the cardia; others (Einhorn, Decker, Dehio and others) are of the opinion that we have to deal with a reflex neurosis, which is caused by mechanical irritations and leads to temporary relaxation of the cardia. According to M. Rosenthal, rumination is brought about by an increased motor irritability of the vagus, whereby the nervus dilatator cardiæ as described by v. Openchowski, causes a dilatation of the cardia opening, and through this an antiperistalsis of the œsophagus is effected. The irritation of the vagus is, according to Rosenthal, very probably of central origin.

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[40] Einhorn, New Yorker medicinische Wochenschrift, May, 1890.

[41] Leva, Münchener medicinische Wochenschrift, 1890, No. 20 and 21.

[42] Freyhan, Deutsche medicinische Wochenschrift, 1891, No. 41.

[43] Decker, Münchener medicinische Wochenschrift, 1892, No. 21.

[44] G. Singer, Wiener medicinische Presse, 1892, No. 45; Deutsches Archiv für klinische Medicin, 1893, Bd. 51, P. 472 (exhaustive description of the modern standpoint on Merycism).

[45] Mayet, Lyon médic., 1893, No. 10.

[46] Linossier and Lémoine, Revue de médec., 1893, Tome 13.

[47] Riesman, The Journal of Nervous and Mental Diseases, June, 1895, P. 359.

[48] Oser, *l. c.*

[Hemmeter[49] reports a very interesting case occurring in his private practice. The case concerns a man of 38 years of age, pianist of exceptional ability. Ordinarily and when in a quiet frame of mind he rarely ruminates, but when he gives instruction, particularly when he has to perform at a concert, or when he is emotionally excited or disturbed, he begins to ruminate (reswallows) for about two hours. In order to stop the ruminating he induces vomiting, which he accomplishes very easily. On one occasion the patient had not ruminated for three weeks, when the time came for him to fulfil an engagement at a concert. The author, for the sake of study, was present when he took his supper on the evening of this concert. Within fifteen minutes after the supper this patient had begun his old bad habit, which kept up during the entire evening, and was plainly observable while he was performing at the piano during the concert.

Is there not a possibility, or rather probability, that the consciousness of the pianist that he was observed by his physician for the very study of this affection acted in reality as suggestion to rumination, especially as Hemmeter in his report considers the affection in this patient as an old bad habit?]

The opinion, which had already been advanced by Luschka and Arnold, namely,. that a dilatation of the lowest portion of the œsophagus in patients suffering from rumination constitutes the cause of the perverse regurgitation, has been elucidated in a very interesting manner by Gustav Singer[50] and v. Hacker. In two of Singer's cases, v. Hacker found a dilatation of the lower third of the œsophagus, and in one of them he observed the mucous membrane at the cardia arranged in the shape of a rosette, a fact which undoubtedly must be attributed to an increase of the transverse section when compared with the normal transverse section. Singer correctly refers this dilatation to the ruminating patient's habit of hasty eating and quick swallowing, whereby a mechanical expansion with following atony of the lowest portion of the œsophagus is brought about.

Diagnosis and Differential Diagnosis.

In cases in which the patients themselves state that they remasticate the food brought up into the mouth, the diagnosis is without difficulties. But frequently patients state only when asked that they chew anew the food brought up into the mouth. On careful consideration of the symptoms, mistaking the condition for vomiting cannot occur. When any

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[49] Hemmeter, Diseases of the Stomach, Philadelphia, 1902, third edition, P. 766.

[50] Singer, *l.c.*

doubt exists, the fact may facilitate the diagnosis that vomiting is alw
preceded by a highly unpleasant preparatory stage (malaise, nau
retching, general depression, pressure in the gastric region); whe
rumination is not at all an uncomfortable condition. The affection b
mistaken for regurgitation could be possible; indeed, both conditions
similar to a certain extent, in so far as rumination is accompanied
regurgitation. In the latter case, however, the food does not come
to the mouth, or if it comes, it is spit out.

As the above-mentioned possibility of a primary organic diseas
the stomach may exist, we will have to examine carefully the gastric c
tents in all cases of rumination accompanied by painful sensations in
stomach.

Treatment.

In beginning rumination there may, under certain circumstances,
some *prophylactic* measures. As rumination is frequently due to ab
mally hasty eating, we must urge the patients to slow swallowing,
care must be taken that the patients do not eat when alone, as then t
would not fear observation. Defects of the teeth require the interv
tion of a dentist. When rumination is well developed, the same m
ures come into consideration, besides the *constant suppression* of the
of regurgitation. That such an auto-suppression can be successf
carried out is proven by cures effected through this measure (Pöns
Einhorn); some cases of mine come into the same class. On the ot
hand, the attempt has been made to cure rumination on the base of
chemical findings of the stomach-contents. Thus good results w
obtained by Alt through administration of alkalies in cases of su
acidity, and by me in a case of subacidity through administration
hydrochloric acid. In treatment of rumination, we will, of course, h
to consider not only the rumination by itself, but also the functions
the stomach, and we must make our conclusions from the findings as
diet and medicinal measures. Besides the auto-suppression, introgas
faradic treatment and strychnia (0.001 to 0.003) by the mouth or hy
dermically in the epigastric region may be tried, with the prospect of s
cess.

Some writers have reported that rumination existed for years wi
out causing any troubles, and that only the attempt of suppressing
act of rumination occasioned gastric disturbances. When rumination i
partial manifestation of an affection of central origin, the latter must
the subject of treatment.

(2) *Regurgitation.*

By regurgitation we understand an occasional and involuntary ascension of food from the stomach into the mouth, from which it is then ejected. The condition falls to a certain degree within the limits of the normal, and only when the ascension of the ingesta occurs to a larger extent and constantly, we may speak of a pathologic condition. Evidently we must deal in regurgitation with a process similar to that in rumination; and, indeed, a habitual regurgitation may gradually develop to rumination. The causes of regurgitation are the same as those of rumination. That just as in the latter, imitation may play an important part is shown by the following case observed by me:—

Mr. S., student of philosophy, 20 years old, American, comes from healthy family. His father, who, as Mr. S. states, is by no means neurasthenic, has the habit of "spitting out" the food after eating. Patient himself has had this affection from his earliest childhood. A quarter to half an hour, particularly after a large meal, the food begins to be brought up again; he usually ejects the regurgitated masses, but reswallows them again when in company. Patient is also capable of regurgitating the ingested food at any minute at will. No subjective or objective symptoms of neurasthenia are present.

In the following case regurgitation developed in a hysteric woman immediately after childbed:—

Mrs. A. F., 36 years of age, was anæmic before marriage, suffered much from hæmicrania; her menses were irregular. The malady began in 1879, a few weeks after giving birth to a child, with regurgitation of swallows of fluids, which patient usually ejected. Temporary improvement alternated with renewed occurrence of this condition, so highly unpleasant to the patient. Appetite rather poor, bowels constipated. Simultaneously with the said neurosis, drawing pains at various parts of the body appeared, which were considered partly as nervous, partly as rheumatic. In 1890 the regurgitation was joined by severe *burning* in the gastric region, which was not relieved by nitrate of silver and iron. (Compare above, p. 649.)

Regurgitation disappeared through Weir Mitchell cure, though the burning returns occasionally.

Peyer (*l.c.*) reports a case, due to sexual neurasthenia, in which regurgitation developed besides other local neuroses.

Diagnosis.

The diagnosis is made from the manner of ascension of food and liquids, and cannot be mistaken for vomiting, owing to the absence of nausea, etc. Whether in the individual cases regurgitation or rumination must be assumed, depends on the occasional or permanent character of the affection [depends upon whether the regurgitated ingesta are or are not remasticated]. It may be considered as undoubted that pronounced rumination may be preceded by a stage of pure or mixed regurgitation.

Treatment.

The treatment of regurgitation is similar to that of rumination. If the affection is caused by neurasthenic or hysteric conditions, these, above all, are to be attacked by various measures (hydropathic measures, change of climate, electrotherapy, etc.). In cases of sexual neurasthenia, the treatment of the sexual apparatus is most important. The direct treatment consists of regulation of diet (slow eating, thorough mastication, watching the patient while eating); furthermore, the consequent suppression of the act of regurgitation; finally, the intragastric faradisation of the cardiac region. In these cases, too, the administration of strychnia might be worth a trial.

(3) *Incontinentia Pylori.*[51]

Preliminary Remarks.—When the stomach is empty the sphincter pylori is usually relaxed, but not during the period of digestion. If it can be proven that it is open also at this stage, we would be justified in speaking of an incontinence of the pylorus. We may assume an incontinence due to either mechanical or nervous conditions. The mechanical form of the pyloric incontinence may occur when the sphincter pylori is destroyed by carcinomatous ulceration, by cicatrised processes after ulcer, or in stenotic conditions in the region of the duodenum. Thus, Stiller mentions a case of compression of the duodenum through the dislocated kidney and consecutive pyloric insufficiency. I have described a case of duodenal stenosis[52] in which the patient died the day after a surgical attempt for removing the stenosis, and in which case an almost complete obliteration of the pylorus was found on post-mortem examination. The neurogenous form of pyloric insufficiency is exclusively supported by

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[51] *Literature.*—Ebstein, Volkmann's Sammlung klinischer Vorträge, No. 155, and Deutsches Archiv für klinische Medicin, Bd. 26; Stiller, Wiener medicinische Wochenschrift, 1879, No. 4 and 5; Müller-Warneck, Berliner klinische Wochenschrift, 1877, No. 30; Oser, Die Neurosen des Magens und ihre Behandlung, 1885, P. 9; M. Rosenthal, Magenneurosen und Magenkatarrh und deren Behandlung, Wien and Leipzig, 1886, P. 92; E. Wilkes, Ueber die Insufficienz des Pylorus, Inaug.-Dissert., Bonn, 1885.

[52] Boas, Deutsche medicinische Wochenschrift, 1891, No. 28.

Ebstein. It concerned, in one of the cases described by that author, a complication of myelitis due to compression; in two others, hysteria, accompanied by tympany of the intestines.

Authoritative writers (Ewald, Einhorn), with whom I agree, consider incontinence of the pylorus as a very rare affection. Einhorn[53] states that the fact that food returns from the duodenum into the stomach is decisive of the diagnosis. If this occur to a greater extent—small amounts are without significance—it would speak for defective closing of the pylorus.

Diagnosis.

The diagnosis of insufficiency of the pylorus rests upon the proof that CO_2 or air introduced into the stomach escapes constantly into the intestine without succeeding in inflating the stomach. Ebstein mentions the following symptoms as diagnostic signs: (1) vomiting ceases which existed before the occurrence of insufficiency; (2) occurrence of intestinal tympany; (3) occurrence of diarrhœa. The last-mentioned signs, however, are, as Oser and Rosenthal correctly emphasise, of doubtful diagnostic value.

According to my experience, the experiment of inflating is decisive only if it always gives the same results; I have repeatedly observed temporary insufficiency of the pylorus in various gastric affections. On the other hand, a paresis of the pylorus may be overlooked when, for instance, it is due to an organic stenosis of the duodenum. In the above-mentioned case of duodenal stenosis I could inflate the stomach *intra vitam* exactly like a normal one. The symptom stated by Einhorn could be of diagnostic value only when simultaneously it would be demonstrated that the stomach could not be inflated by air introduced from without.

[Hemmeter[54] recommends for the diagnosis of insufficiency of the pylorus his method of intubating the duodenum, or the use of the spiral revolving sound of F. Kuhn and F. B. Turck for sounding the pylorus. Hemmeter (*l.c.*) quotes an interesting observation of a case of insufficiency reported by Schütz,[55] in which it was possible to distend the stomach by means of CO_2, but not by air. The increased irritation of the mucous membrane of the pylorus by the carbonic acid gas caused a contraction of the annular muscle, but with the forcing in of air the stomach did not become distended, and this points to the fact that different degrees of insufficiency occur.]

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[53] Einhorn, Die Krankheiten des Magens, Berlin, 1898, P. 319.

[54] Hemmeter, Diseases of the Stomach, Philadelphia, 1903, third edition, P. 56 and 770; Johns Hopkins Medical Bulletin, April, 1895; Archiv für Verdauungskrankheiten, Bd. ii, P. 85.

[55] Schütz, Prager med. Wochenschrift, 1882, No. 11.

Treatment.

A treatment for incontinence of the pylorus does not exist; for purpose of a cure we ought to remove the cause of the insufficiency. Einhorn saw good results in a case assumed as pyloric insufficiency, from intragastric faradisation and lavage. The mechanical form of pyloric insufficiency is, to a certain extent, a curative process, the removal of which is consequently not indicated. [Hemmeter advises a particularly careful diet, massage, internal and external galvanisation and faradisation applied directly to the pylorus by his method, douches, and eventually also by giving strychnine (0.002 three times a day). In some cases he recommends dilute hydrochloric acid.]

(4) *Nervous Eructation.*

(Eructatio Nervosa.)

Preliminary Remarks.—Nervous eructation is manifested by the fact that, independently from the meals, paroxysmal explosive eructations occur. There are periods in which belching ceases entirely, only to be incited again after some time by known or unknown causes. In general, we assume that in cases of nervous eructation we have to deal with swallowed air, which is evacuated from time to time through antiperistaltic movements of the stomach. According to Oser (*l.c.*), we have to deal with aspiration of air; he compares the stomach to an elastic balloon attached at the end of the œsophagus. As soon as the circular muscle fibres contract, air is expelled; and as soon as they relax, air is sucked into the stomach anew from the œsophagus. Bouveret[56] does not agree with this view. He believes there is a very active circulation of air from the mouth to the œsophagus, whereby occasionally, but not always, air may also enter the stomach. He considers the real cause for the circulation of the air to be a spasm of the pharynx, whereby a spasmodic deglutition is occasioned and the aspirated air escapes.

Nervous eructation is particularly frequently observed in hysteric women, although there are attacks of this kind in some cases of neurasthenic men. I especially remember a very troublesome form of eructatio nervosa in a dentist, which refused to be relieved by any remedial measure. A small lipomatous hernia was discovered by somebody else; after an operation the affliction ceased immediately. The causes are usually fright, anger, losses, perhaps also hasty eating. Peyer mentions also cases of nervous eructation due to sexual neuroses. [I had under my observation a woman who was afflicted with most troublesome eructations, lasting day and night. The primary cause was general neurasthenia, caused probably by marital troubles (divorce)—or were the marital troubles caused by the neurasthenia? Patient had general enteroptosis, fibroma in the uterus, and was of very erotic nature. After patient had, under special diet, gained about thirty pounds in weight, eructation ceased along with the disappearance of other neurasthenic symptoms.] The gases produced by the eructations are odourless, and consist principally of atmospheric air.

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[56] Bouveret, Les maladies de l'estomac, 1893, P. 609.

Some time ago I had occasion to observe a case of nervous eructation probably due to "infection."

Mrs. M., farmer's wife, aged 45 years. Mother and an aunt (father's sister) suffered from the same affection, from which patient is now suffering. The trouble has lasted as long as she can remember. It appears paroxysmally (about every week), lasting several (three or four) days, and disappears gradually. There develop spasmodic bellowing (belching) eructations, which follow one another ten to twenty times at intervals of five to ten minutes. Before eructation, marked pressure; afterward, relief. In the intervals between the periodical attacks there exists often loss of appetite; pressure after meals. Otherwise patient had never been ill. The affection is essentially exacerbated, or even brought about by excitement. During night time the attacks usually cease, but there are occasional exceptions.

Diagnosis.

The diagnosis is not difficult as soon as one has occasion to observe a paroxysm of eructation, and simultaneously considers the absence of any signs of an organic gastric affection (particularly also absence of objective distension of the stomach). As shown by a case from Kussmaul's clinic, reported by Pönsgen, paroxysmal eructation may occur also in carcinomatous stenosis of the pylorus; the examination of the stomach-contents in doubtful case is, therefore, very important. In all cases which were examined by me I found normal conditions of hydrochloric acid and motility. Of diagnostic significance might also be the determination of the quality of the gases according to G. Hoppe-Seyler's method.[57] The detection of atmospheric air would speak in favour of nervous origin; that of hydrogen, carbonic acid, or nitrogen, in favour of fermentative products.

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[57] G. Hoppe-Seyler, Verhandl. des Kong. f. innere Medicin, 1892, P. 293; Deutsches Arch. f. klin. Med., Bd. 50, P. 82.

Treatment.

The treatment must, above all, consider the neurasthenic basis and the ætiology of the affection. Change of climate (mountain, sea-shore) is usually of excellent influence; likewise hydropathic measures are to be tried. In medicinal respect, I prefer particularly the bromides in small doses, as recommended by Oser. I have not enough experience with the other medicines, recommended also by Oser, such as arsenic, extract of belladonna (or atropine), or chloral hydrate. Sometimes the troublesome affection disappears without any treatment, but only to return again.

(5) *Pneumatosis.*

(Flatulent Distension.)

By pneumatosis we understand a condition in which the stomach is markedly expanded with air, whereby a sensation of unpleasant distension, dyspnœa (asthma dyspepticum, Henoch), precordial oppression, etc., may be occasioned. When the air escapes upwards through the cardia or downwards through the pylorus, the torturing feeling ceases. The suffering usually occurs periodically, sometimes without evident cause, at other times subsequent to psychic emotions. I observed pronounced cases only in men. In all my observations the functions of the stomach were intact, and the ingestion of food does not have any influence whatever upon the occurrence of the paroxysms. A spasmodic closure of the sphincters pylori and cardiæ has been assumed in order to explain the condition. The suffering may be permanent or periodical, appear and disappear through external influences.

Pneumatosis occurs as an idiopathic neurosis or as chief symptom of general neurasthenia, but also as a more or less pronounced concomitant symptom of other gastro-enteric affections. Thus, an abnormal collection of air occurs very often in cases of atony and dilatation of the stomach. Only the first-mentioned type is to be accepted as pneumatosis in the stricter sense.

Diagnosis and Differential Diagnosis.

The diagnosis is made partly from the statements of the patient, partly from the observation of the stomach itself during the paroxysms, when the organ is found to be distended like an air-cushion; the respiration, as well as the pulse, may be exceedingly accelerated or irregular.

We must consider whether we have to deal with the stomach, or perhaps with a distended transverse colon. In a number of my cases there existed the above-described symptom-complex of pneumatosis; but it was evidently caused by a more or less pronounced coprostasis; after its removal the abnormal collection of air disappeared. The presence of an organic gastric affection, particularly of a gastric dilatation and atony, must be excluded, which, in most cases, will not be difficult. In atony there occur, as I reported,[58] in rare cases conditions of of asthma dyspepticum, which, however, distinguish themselves essentially from pneumatosis through the course, especially the vomiting. Furthermore, we must guard against mistaking pneumatosis for certain forms of angina pectoris. In these cases there is a *pain* located in the scrobiculus cordis and radiating toward the sternum and either side, which disappears very soon when the patient is in a recumbent position or is standing quietly, of course, often with eructation of air.

Treatment.

The state of the general nervous system must be improved by application of hydropathic measures, change of climate, avoidance of psychic emotions, etc. As to medicinal treatment, nervina, bromides, cocaine, and morphine may be considered. In a case of Ewald[59] hypodermic injections of morphine were accompanied by good results. In some cases of gastro-enteric pneumatosis I obtained good results from a combination of extract of calabar bean and extract of nux vomica, according to the following formula:—

℞ Extracti physostigmatis	0.5
Extracti nucis vomicæ	1.0
Succi et pulveris liquiritiæ	q. s.

ut fiant pilulæ No. L.
Sig.: One pill three times a day.

(6) *Peristaltic Unrest (Kussmaul[60]).*

(Tormina Ventriculi Nervosa—Gastric Hyperperistalsis.)

While under normal conditions, even with increased digestive activity, visible or palpable peristaltic movements of the gastro-enteric canal are absent, under pathologic conditions peristaltic actions of varying duration and extent may occur in the alimentary tract. Sometimes this peristaltic unrest may concern particularly the stomach, at other times the intestine; or stomach and intestine may exhibit this peculiar condition simultaneously. These abnormal peristaltic movements may be directly felt as "undulations and contractions in the abdomen" (Kussmaul), and may appear at any time, whether the stomach is full or empty, by day or night. The ætiological factor of this abnormally increased motor function may consist either of a mechanic cause (pyloric stenosis, carcinoma at or in the neighbourhood of the pylorus), or of a peculiar motor neurosis, for which Kussmaul has contributed interesting observations. Peristaltic motions can be seen and felt likewise in the region of the intestines when the passage has become difficult in any way, through partial dilatation, or if irritative conditions of the intestinal mucosa or abnormal fermentations are present; but here also there occur purely nervous forms of excessive peristalsis.

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[58] Boas, Archiv für Verdauungskrankheiten, 1896, Bd. 2, P. 444.

[59] Ewald, Klinik der Verdauungskrankheiten, Bd. 2, P. 428.

[60] Kussmaul, Die peristaltische Unruhe des Magens, etc., Volkmann's Sammlung klinischer Vorträge, 1880, Nó. 181.

In the following I mention briefly an interesting case of this kind of tormina intestinorum nervosa:—

Miss Martha F., 22 years old, family healthy; patient always well except habitual headaches. Has been ill for three previous weeks, as she states as result of severe excitement. The affection is manifested as follows: independently of ingestion of food or other conditions, there appear at daytime, particularly in the afternoon, pains in the abdomen, beginning on either side and drawing towards the front; after a short time a gurgling noise becomes distinctly audible and the pain ceases, only to begin anew a few minutes later. During the paroxysm of pain the abdomen is markedly distended. Appetite is poor since beginning of affection; there is frequently pressure in the region of the stomach, nausea and vomiting; bowels constipated.

Status præsens: abdomen is hemispherically distended, corresponding to pregnancy of the eighth month. All over the abdomen loud, tympanitic sound. At various places undulatory contractions are seen and felt, especially to the left and below the umbilicus, which last for a few seconds, disappear, and continue towards the right. Simultaneously with the relaxation of the area concerned a loud gurgling is heard just like the pushing of fluid into a narrow cylinder. In the intervals, on palpation of stomach, nothing abnormal is found, especially no symptoms of ectasia. The manner of occurrence and the outlines of the contracted area certainly prove the intestine as the seat of affection. Codeine and belladonna, as well as faradisation of the abdominal walls, were used, with permanent effect.

Besides the excessive *peristaltic* motions, there may, as Schütz,[61] Cahn,[62] and Glax[63] have shown, appear also *antiperistaltic* motions. In these cases the waves run from right to left, while normally they run from left to right. In Cahn's case peristaltic and antiperistaltic motions could be seen simultaneously.

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[61] Schütz, Prager medic. Wochenschrift, 1882, No. 11.

[62] Cahn, Deutsches Archiv f. klin. Medicin, 1884, Bd. 35, P. 402.

[63] Glax, Pester medicin-chirurg. Presse, 1884.

Diagnosis.

The diagnosis, as far as it concerns the fact of peristaltic unrest, is easily made by inspection. The ætiology of the phenomenon is more difficult to discover. An exact physical and chemical examination will give information in the majority of cases, whether we are dealing with a mechanic obstacle or with Kussmaul's type of tormina nervosa. The state of the general nervous system must be thoroughly considered.

Treatment.

The treatment must vary according to the ætiology of the affection. In the purely nervous type of the peristaltic unrest the general nervous system must be strengthened by climatic and hydrotherapeutic measures. Kussmaul observed in the above-mentioned cases a favourable influence from the internal and external application of strong faradic currents.

Rosenthal,[64] in a case of peristaltic unrest, accompanied by constipation, obtained good results from the administration of larger doses of bromides and extract of belladonna. In the case mentioned above I observed the attacks cease after administration of codeine and belladonna and the application of electricity to the abdominal walls. In a second case of mine the attacks ceased when the patient was admitted to my private clinic for closer observation.[65]

(7) *Cramp of the Cardia.*

(Cardiospasm, Spasmodic Dysphagia.)

By cardiospasm we mean a transient condition of contraction of the lowest portion of the œsophagus associated with spasmodic pain in the cardiac region, not dependent upon any anatomic changes. The ingesta are masticated without difficulty and reach the œsophagus. In the lower portion of the œsophagus there begins a disturbance of deglutition: the food is brought up again with more or less exertion immediately or after some time, only a small part of the food reaching the stomach. As result of this, and through mechanic disturbances due to the obstruction in the œsophagus, the state of nutrition may suffer severely, even so severely that, as a case described by v. Monakow[66] teaches, the fatal ending could not be avoided.

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[64] Rosenthal, Magenneurosen und Magenkatarrh, Wien and Leipzig, 1886.

[65] [Boas-Basch, Diseases of the Intestines, New York, 1901, English edition, P. 524 *et seq.*]

A slight transient spasmodic narrowing of the cardia may occur physiologically, for instance, on introduction of the stomach tube. Furthermore, cardiospasm may occur as a transient and less serious pathologic process, or as a chronic and grave process. Ætiologically it is not always easy to elicit whether a peripheral (ulcer, superacidity, slight abrasions) or a central irritative process is present.

Diagnosis.

The transient and paroxysmal cardiospasm is characterised through *painful cramps* in the cardiac region, which disappear immediately after the state of contraction has passed, whether the inciting cause was natural or artificial. The affection is rather easily recognised as a neurosis through its periodic occurrence, and the seat of the affection is easily elicited in the majority of cases through the peculiar manner of occurrence.

Much greater difficulties are found in the second type of cardiospasm, the *chronic* cardiospasm. There may exist occasionally normal deglutition, but in the majority of cases serious troubles develop, which are principally caused by the organic changes that are brought about gradually by the affection, which in the beginning was a purely nervous spasm.

As the following case may perhaps give many a hint for the diagnosis, we will report it, especially as cases of this kind are not overfrequent, more in detail than the other histories:—

Mr. A. H., from Buenos Ayres, 32 years old, comes from healthy family, but mother is somewhat nervous, on account of uterine trouble. Patient was always well. In 1880 he went to Buenos Ayres, where he lived a restless life, irregular in his eating and drinking. In March, 1885, patient underwent especially severe business excitement, which was followed in the beginning by *crampy pains, appearing about three or four hours after each meal, and radiating towards back and chest.* They were, however, but transitory. Various remedial measures were without effect. These pains were accompanied by a certain "feeling of anxiety" [oppression] in the lower portion of the œsophagus, associated with salivation. After a while patient noticed that the passage of food met with difficulties, in as far as he succeeded in overcoming the same only by pressing, swallowing, and drinking of water. At that time dilatation of the stomach was diagnosticated by the physician, the patient was confined to a strict diet, lavage of the stomach was applied; the effect obtained was but transitory.

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[66] v. Monakow, Korrespondenzblatt für Schweizer Aerzte, 1893, P. 310.

In 1887, patient consulted Professor L., in Würzburg. A definite diagnosis was not made, as patient states, but gastric dilatation was excluded; patient was advised to introduce a thin bougie. As a whole, patient felt much better while residing in Germany. Already on the return-journey the condition deteriorated in spite of the use of the bougie; patient lost weight, and he was compelled to leave Buenos Ayres and to stay in the country. In 1890, patient, who was very much emaciated, again went to Professor L. At that time a diagnosis of diverticle was made, and patient advised to be fed entirely through the tube. After this treatment also had been fruitless, patient consulted Professor E., in Heidelberg, whose opinion was that it concerned a weakness of the gastric musculature, and that the "diverticle" was not secondary (?). He advised rest and but moderate use of the bougie. The question of surgical interference came up, but was very soon dropped as impossible. In March, 1891, patient returned to Buenos Ayres, and introduced *pretty thick* bougies into the stomach, whereby his condition improved. But at each meal large amounts of food remained lodged above the stomach. Patient temporarily gained some weight, but, as a whole, his condition remained the same. In October, 1891, patient came under my observation.

Status præsens: at the sternal region everywhere full sound. Heart-sounds clear and distinct; radial pulse, equal on either side. Stomach is slightly atonic, the greater curvature on a full stomach about two centimetres below umbilicus; nowhere tenderness on pressure. In introducing the œsophageal tube one encounters, at a distance of 35 centimetres from anterior incisors, a resistance which at first cannot be overcome. At the same time, patient expresses large quantities (half a litre) of undigested liquid food. He succeeds easily in doing so when he bends forward. The liquid masses, which, on repeated introduction of the tube, were always examined as to their reaction, react, evidently according to the nature of the ingesta and the time of their retention above the stomach, sometimes slightly acid, at other times neutral, rarely alkaline. But free acidity never could be detected. On microscopic examination it was found that the masses were turgid, slightly changed food-remnants.

After removal of these quasi barricading masses, the tube readily passed into the stomach, and now, if the stomach was full, a normal chyme with HCl reaction could be extracted. The amount of hydrochloric acid was in normal limits. Patient had made use of this experience, and removed, before every mealtime, the retained ingesta through the tube introduced as described above.

The examination of the deglutition sounds exhibited a distinct first sound; the second, however, varied; at times several minutes after deglutition a rippling noise could be heard, but not the normal sound; usually it was entirely lacking.

In consideration of all conditions involved, the diagnosis wavered between a deep-seated diverticle—to be sure, a rare occurrence—and cardiospasm. Patient himself decided the differential diagnosis. He made the observation that he was able to eat a large meal, consisting chiefly of solid food, *without retention of substances being detected on subsequent examination by means of the tube.* After the patient, with my consent, in the following days had ingested similar meals with similar results, the diagnosis was decided; undoubtedly there was a temporary spasm of the cardia, or, perhaps more correctly, of the subcardial portion of the fundus of the stomach. From this moment I forbade soup diet entirely, advised him to live in his usual manner, and before each meal to introduce a thick tube and to let it remain there for a few minutes. Simultaneously small doses of ammonium and sodium bromide were given by the mouth, and intraventricular applications of weak galvanic currents were used. The result was excellent; the patient could eat any kind of food, took his meals at the table d'hôte, a thing he had not been able to do for five years, and recovered visibly. From that moment only small amounts of remnants, or nothing at all, could be removed from the œsophagus on introduction of the soft tube, and the same was also empty in the morning.

The diagnosis of spastic cardiac contraction could be directly demonstrated a few times, for when very thin Nélaton catheters were introduced, it was, though the œsophagus was entirely empty, impossible to push the same past the cardia. Only after the thick catheter had been passed through the cardia and left there for a few minutes, the thin catheter also easily entered the stomach. It was remarkable, also, that patient, after change of diet, gained sixteen pounds in weight within two weeks. Later there developed a permanent dilatation of the œsophagus. Netter,[67] who reported the case from Fleiner's clinic, assumes as its cause a primary relaxation of the œsophagus. But, as shown by the previous history (see above), the first symptoms were those of a true cardiospasm, which was followed later, similarly to cases of spastic pyloric stenosis, by manifestation of retention above the contracted portion.

Differential Diagnosis.

The diagnosis of a spastic cardiac closure encounters hardly any difficulties in cases in which the spasm is temporary. But if there exists a permanent spasm, the diagnosis will frequently be undecided between ulcer of the œsophagus, carcinoma of the cardia, deep-seated diverticle, and cardiospasm. As to the differentiation from peptic ulcer of the œsophagus, we must consider pain regularly occurring in the course of the œsophagus or at the cardia at the time of ingestion of food. If it is associated with hæmatemesis—it will be difficult to distinguish between bleeding from the œsophagus and that from the stomach—it would speak with great probability in favour of peptic ulcer of the œsophagus. Rosenheim[68] recommends as a most decisive means of differentiation the examination through the œsophagoscope. [Compare Professor Dr. Hugo Starck, Die Direkte Besichtigung der Speiseröhre, Œsophagoskopie, Würzburg, 1905.]

In regard to other affections in question, we give a table of the most important diagnostic symptoms:—

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[67] Netter, Archiv für Verdauungskrankheiten, 1898, Bd. 4, P. 114.

[68] Rosenheim, Pathologie und Therapie der Krankheiten der Speiseröhre und des Magens, 1896, 2 Auflage, S. 136.

	Cardiac Carcinoma.	*Deep-seated Diverticle.*	*Cardiospasm.*	*Dilatation.*
Age:	Advanced age.	Independent from age.	Independent from age.	Independent from age.
Examination with Bougie or Tube:	Frequently traces of blood and pus, in rare cases particles of the tumour. Thinner bougies or tubes pass easier than thicker ones. Sometimes small amounts of food remnants.	Two tubes are introduced; one (*a*), with lateral openings up to a distance of 30 centimetres from the teeth, is pushed till in the stomach; the other (*b*) remains in the œsophagus. If water is introduced through tube *b*, it cannot be aspirated through tube *a*, but through tube *b* (Rumpel's experiment). No blood or pus in the œsophageal contents.	Thicker bougies or tubes pass easier than thin ones. A tube with lateral openings is passed till it reaches the stenosis. If now water is introduced, and auscultation is performed in the stomach region, the tube will enter the stomach (Kelling's experiment.) No food-remnants, no blood or pus.	In making Rumpel's experiment (*vide* diverticle), the water introduced through tube *b* may be syphoned out or aspirated through tube *a*, but not through tube *b*. No blood or pus in œsophageal remnants.
Gastric Contents:	Usually no free HCl.	Normal or superacid.	Normal or superacid.	Normal.

Through introduction of a tube into the diverticle, and of another tube into the stomach and washing out with coloured fluids, the diagnosis of diverticle may be made more certain. Through Röntgen rays, diagnosis can be ascertained by filling the diverticle with bismuth.

It must be added that cardiospasm not infrequently develops as a result of neuropathic conditions, so that the detection of other nervous symptoms would speak in favour of spastic origin of the cardiac closure. As shown by the history described above, real dilatation may develop from spasms of the cardia or the lowest œsophageal portion when they have lasted many years.

Treatment.

Besides the rational general treatment to which belongs a roborating, by no means only liquid diet, the affection requires local treatment by means of bougies. Bougies as thick as possible are passed *into the stomach* and permitted to remain for a long time. Thin bougies and elastic tubes do not fulfil the purpose. Of internal remedies, there are, above all, bromides in large doses (5 to 8 grammes pro die), and also cocaine, morphine, and codeine, the effect of which is uncertain.

In the above-mentioned case I applied weak galvanic currents to the upper fundus region, after introducing an electric tube into the stomach filled with water. How far this application aided the favourable course it is difficult to state; nevertheless, the intragastric galvanisation of the pars cardiaca ventriculi deserves consideration along with the other curative methods. Where abnormal accumulation of air leads to the symptoms of cardiospasm, the air is to be removed by methodic introduction of the tube, and the patients are told to avoid the supernormal swallowing of air. In the above-mentioned cases of v. Monakow, hypnotic suggestive treatment was applied without effect, but chloroform narcosis was accompanied by transient effect.

(8) *Nervous Vomiting.*

(Vomitus Nervosus, [Habitual or Reflex Vomiting].)

Preliminary Remarks.—We mean by nervous vomiting only those forms of vomiting in which the gastric mucosa itself is intact and the stomach represents but the intermediate organ for the act of vomiting. The vomiting may be of cerebral or spinal origin, or it may be reflex vomiting, arising from various organs (genitals, pharynx and larynx, nasal mucosa, kidneys, liver, spleen, peritoneum, cæcum, and others), or may be a concomitant symptom of neurasthenia and hysteria. Special consideration must be given to *juvenile vomiting, gastric crises,* and the so-called *periodic vomiting.* The subordination of certain types of vomiting under one of these classes is not only important for a perfect diagnosis, but offers also important hints for their treatment.

Diagnosis.

The diagnosis must be divided into two parts: first, to ascertain as much as possible the nervous origin of the affection; and second, to elicit the ætiology. The first part may encounter difficulties. Stiller, in his excellent book on the nervous gastric diseases, gives a record of those points which are to be considered as to the nervous origin of the vomiting, as follows:—

1. Facility of vomiting.

2. Independence of vomiting from quality and quantity of ingesta in general.

3. Freakishness with which certain frequently very odd nutriments and refreshments are retained exclusively.

4. Occasional selective vomiting of certain substances, which are even separated from the mixed chyme.

5. Carelessness with which the patients usually endure the habitual affection.

6. The astonishing tolerance of the body to the effect of inanition caused by the habitual vomiting, even when the metabolism is much reduced.

7. The extraordinary influence of the slightest external and internal conditions, which act on mood or temperament.

8. The frequent occurrence of vomiting when no food has been ingested and the stomach is apparently empty.

9. The presence of other nervous symptoms synchronous or alternating with vomiting.

We may add another point:—

10. The absence of important secretory or motor disturbances of the stomach.

While with careful consideration of these symptoms the nervous origin of the vomiting may usually be recognised, the question of ætiology is more difficult, because the ensemble is not infrequently obscured through a series of concomitant symptoms. Especially the differentiation between a functional neurosis and a central affection is frequently made possible only by most careful observation during some length of time. Under no circumstances must we neglect in doubtful cases to examine for papillitis as well as for albumen in the urine, and an examination of the genitals (particularly in the female) must never be omitted.

(*a*) *Juvenile Vomiting.*—In school-children there develop often conditions of dyspepsia, cardialgia, and vomiting as a result of mental overexertion. The latter is frequently habitual, daily or almost daily, but in other cases it shows a purely periodic character. M. Rosenthal[69] furthermore mentions, besides the dyspeptic troubles, cardialgia and vomiting as symptoms common to four similar cases, great pallor, slowing of the pulse down to sixty, dilatation of pupils. In all these cases permanent improvement followed only after the children were temporarily removed from school and after roborating care and diet. v. Leyden[70] also observed cases of juvenile vomiting in mentally overburdened children. I regard the nervous "school vomiting" as an affection by no means rare, considering my numerous observations.

The *diagnosis* is conditioned by the consideration of age, appearance, and the above-mentioned symptoms. To be sure, we must not forget that the cause of the vomiting may rest in an organic central affection. An exact analysis of the symptoms and observation for some length of time will probably lead to the correct diagnosis in the majority of cases.

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[69] M. Rosenthal, Magenneurosen und Magenkatarrh und deren Behandlung, Wien and Leipzig, 1886.

[70] v. Leyden, Zeitschrift für klin. Medicin, 1882, Bd. 4, P. 605.

(*b*) *Crises Gastriques, Gastric Crises.*—To Charcot is due the great merit of having first classically described this peculiar complication of tabes dorsalis. There are typical and atypical forms of the gastric crises. The former are distinguished by the fact that there appear suddenly during perfect well-being severe cramp-like pains radiating towards abdomen and back, which are followed by copious vomiting. In the beginning food is vomited; later, mucus, bile, and duodenal juice. In rare cases (Fournier,[71] Charcot[72]) there may occur hæmatemesis, which may bring diagnostic doubts. The vomiting occurs during day-time repeatedly, often every hour or still more frequently. The patient is immediately confined to bed, there is excessive weakness, appetite is lost entirely, but there exists great thirst. The urine is scanty and light of colour, bowels are constipated, the abdomen is much retracted, the pulse is small and soft. As a whole, the patient presents a picture of severe collapse. During the night the spasmodic vomiting usually ceases, to return again on awakening. Apart from this interruption the attack may last for two to eight days, or even longer. The vomiting may cease just as rapidly as it came; a feeling of hunger returns in the highly debilitated patient, and he can eat and endure again all kinds of food. There are, however, deviations from this typical course, as, according to observations of v. Leyden,[73] vomiting in gastric crises may be very insignificant or may be entirely absent. On the other hand, according to my experience with two cases of tabes, the pain may be but slight. In some cases there develops, according to my observations, a condition of chronic crises which may last for weeks and months. Finally, the attacks are not to be referred to the stomach, but to the intestines; they are designated as *crises entériques* (intestinal crises).

The *diagnosis* is readily made when manifest symptoms of locomotor ataxia are present; but, as mentioned above, the gastric crises are often one of the earliest symptoms. *In every case of periodic vomiting the reaction of the pupils, tendon reflexes, and the sensibility are to be tested.* But even if these symptoms should be normal, the prognosis of periodic vomiting ought to be made cautiously, as observations of Charcot[74] have shown, because the classic symptoms of tabes may make their appearance many years after the first occurrence of the crises.

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[71] Fournier, Leçons sur la période préataxique du tabes d'origine syphilitique, Paris, 1885.

[72] Charcot, Gaz. médec. de Paris, September 28, 1889.

[73] v. Leyden, Verhandlungen der Gesellschaft der Charitéärzte, Berliner klin. Wochenschrift, 1888, No. 53.

[74] Charcot, *l.c.*

(c) *Periodic Vomiting.*—In otherwise healthy persons there develop sometimes, usually in definite intervals (of two, four, eight, twelve weeks, etc.), attacks of vomiting which very much resemble the clinical picture of the gastric crises. The attacks, which were first classically described by v. Leyden,[75] begin either with certain prodromal symptoms (headache, sensation of weakness, lassitude, etc.), or begin in the midst of apparent good health. The paroxysm starts either exactly analogous to the typic form of gastric crises, with severe gastric pain, radiating to the sides and back, which is followed by vomiting, or vomiting introduces the attack.

First food, later mucus, finally bile and duodenal juice are vomited. The clinical picture is very similar to that in gastric crises [see above]. The condition in severe cases may last from one to fourteen days; at night the paroxysm is usually interrupted by sound sleep. Quite suddenly normal appetite and tolerance for all kinds of food return, so that the patient, though still weak and debilitated, is able to leave the bed. His appearance gains the former freshness, the body the normal tension, bowels and urine secretion become regular.

If we would content ourselves with the *diagnosis* of periodic vomiting itself, the condition would be, in almost all cases, readily recognised, from the clinical picture first described. But there are other gastric affections which may present similar pictures. To these belong:—

1. The above-mentioned gastric crises. Of differential importance is, as I have emphasised,[76] the fact that the gastric crises show an atypic occurrence, namely, that they may occur in irregular intervals of weeks and months, while in the so-called idiopathic vomiting a certain typic periodicity is undoubted. It is true, no positive importance can be attributed to this symptom.

2. Periodic supersecretion (Riegel, Jaworski, Reichmann, and others) may be readily detected by means of the stomach tube. Gastroxynsis also may be easily differentiated from periodic vomiting, through the severe headache introducing the former and the vomiting of intensely sour masses; after the vomiting the attack usually ceases.

3. Malaria dyspepsia may be thought of, but in these cases the vomiting is not so pronounced; the symptoms point more to an organic gastric affection. An enlargement of the spleen, which would speak in favour of malaria, may be absent (M. Rosenthal, v. Leube).

[Hemmeter[77] quotes in his text-book the report of a case of periodical hæmatemesis occurring every third day, which was cured by quinine. In gastric trouble showing any periodicity, the blood ought to be examined for the presence of the malarial parasite.]

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[75] v. Leyden, *l.c.*

[76] Boas, Deutsche medicinische Wochenschrift, 1889, No. 42.

[77] Hemmeter, Diseases of the Stomach, third edition, P. 386.

The real cause of the suffering ought to be determined in order to derive some prognostic and therapeutic hints. Although the disease may occur as an idiopathic neurosis (vagus neurosis, v. Leyden), palpable causes may be existing for the periodic vomiting, as, for instance, dislocated kidneys, hydronephroses, uterine and ovarian affections, entozoa, nicotinism. If a special ætiology is not evident, the prognosis must be made very cautiously, in consideration of a possible incipient or later-developing spinal disease, particularly tabes.

[The following case of gastric crises, which has been under my observation for over six years, is worth describing here, not only for the fact that for a number of years it apparently has been the first and only symptom of tabes dorsalis, but also as to the treatment administered in the case, which was undoubtedly of excellent effect upon the gastric crises:—

[J. F., painter, born 1847, in Philadelphia. His father, a fireman, hard drinker, died when 46 years of age, of consumption. Mother had womb-trouble, 60 years of age, died of a hæmorrhage while sitting in a chair, after having been afflicted with a "cold." A brother died, when about 60 years old, of complaint similar to what patient has. Another brother died of heart-trouble and dropsy, when 64 years old. A third brother died in childhood. A sister died when young. Patient is the youngest child. Mother's sister died of heart-trouble and dropsy.

Patient came under my observation in the Philadelphia Polyclinic Hospital in summer of 1900.

History.—Patient was healthy when a child, except measles. Had gonorrhœa several times before he married, at the age of twenty-one. His wife is healthy, had three children, one child one year after marriage, and twins seven years after marriage. One of the twins died of pneumonia at the age of four years. No miscarriages. Patient, at the age of 34, had an ulcer on the penis, which took about five to six weeks to heal; he had sore tongue and sore throat, hair fell out somewhat, no rash—he took medicine, never had inunctions. Patient felt pretty well till fifteen years ago. Had pain in the shoulder—sometimes right, sometimes left—lasting, in the beginning, only a second—there was a kind of stinging pain—gradually getting worse and more painful, lasting a day or two. Then he vomited—no real pain in the stomach—a pint at times, every half to one hour; continued sometimes for three or four days; remained in bed. The vomit had plenty of mucus and bile; tasted more bitter than sour. Sometimes the attack lasted a whole week, could not eat, felt miserable, weak, dizzy—had several fainting spells, once in his physician's office. After the attack could eat, drink, and smoke (cigars and pipe); was rather a heavy drinker (five to six whiskeys, even up to ten to fifteen, much beer, "plenty drunk"). He felt all right till a renewed attack came at intervals of three or four to six months.

He stated that he had been under treatment by a number of physicians and in various hospital dispensaries, but had not received any marked relief.

At the time of his first visit to the Polyclinic he was examined by Dr. E. B. Sharp, of Camden, N. J., and myself, assistants to Dr. D. D. Stewart at the Department for Diseases of the Stomach and Intestines.

Patient, 53 years of age, 165 centimetres tall, weighs 115 pounds, emaciated, but not very weak. Chief complaint, attacks of pain in the stomach and back, and vomiting of sour liquid masses. In the intervals between the attacks feels pretty well and very cheerful.

Examination shows: lungs normal, heart slightly irregular, liver somewhat enlarged; no pain on pressure on stomach or intestines. Left pupil very much contracted, right one less contracted; knee-jerks lost; very slight ataxia; co-ordination fairly good; dizziness on closing eyes; sensation of temperature and muscle-tonus normal; no anæsthesia; sensation of belt around the body not present. His bowels are constipated. Urine at times scanty, but by no means always so; albumen present; no sugar; an occasional hyaline cast. Oral cavity does not show anything abnormal; no blue (lead) seam on the gums.

Stomach examination shows a slight dilatation, but fairly good motor function. Various test-meals always yield a high degree of hydrochloric acid; no fermentation acids.

Treatment (general): inunction with mercury (vasogen-hydrargyrum ointment) and potassium iodide. Local, Priessnitz's Umschläge (cold wet pack) over night, lavage of the stomach with nitrate of silver solution (1 to 1000), followed by bismuth. The amount of hydrochloric acid decreased markedly. For the pain I administered occasional doses of cocaine hydrochlorate in capsules. Patient came to my private office in March, 1901, and has been under my observation since. In other cases of locomotor ataxia I had found pills of silver nitrate (argent. nitr. 0.25, argill. alb. q. s., ut fiant pilul. No. xxv, one pill evening and morning) of good service, and I administered these also in this patient. Patient, for the last four years, has had decided relief through the treatment; attacks are slighter, and sometimes do not return for a year; bowels move regularly every day. Patient has gained as much as 34½ pounds in weight.

In the last two years, however, he showed decided signs of lost compensation of the heart, and disturbances in the portal vein system; at various times ascites and anasarca showed themselves, which, however, readily disappeared after calomel and digitalis. On one occasion the ascites was of such a degree that within thirty hours twenty-four quarts of urine passed, besides the water evacuated with the stool.

Patient, at the present time, in a fair condition, and has frequently worked at his usual occupation, house-painting.]

Treatment.

The treatment of *vomitus nervosus* must be directed principally to the underlying cause. Only when the cause remains obscure we have to content ourselves with symptomatic treatment. Its main principles are rest and avoidance of external, and particularly psychical influences; under these circumstances isolation of the patient or change of climate is frequently of greatest importance, especially in the young. Dietetically, it must be remembered that the more fluid is contained in the stomach, the easier the stomach is emptied. The fluid should therefore be limited, and should rather be replaced by easily assimilable solid substances; of course, these also only in small quantities, as, for instance, cakes [crackers, biscuits], zwieback, scraped beef, soft-boiled eggs, rice, farina, noodles, and similar food. Ice-pills have an important sedative action. [I find that usually ice increases thirst, which results in the desire for more ice; and the effect is frequently the reverse of what is expected; the patient vomits more frequently. If thirst is present, rinsing the mouth with hot water allays the thirst and removes irritability of the patient.] The sedative effect of ice may be enhanced by the addition of tincture of belladonna (ten to fifteen drops every three or four hours), or of laurel water (aqua laurocerasi), and also of chloroform (three to five drops). According to the tolerance of the stomach, selection and quantity of the food may be changed every week, but always with the greatest caution. A certain cautious experimenting can hardly be omitted.

When abnormal hyperæsthesia of the stomach is present, the feeding by the stomach may be temporarily (a week or ten days) substituted by rectal feeding, according to the directions recommended in the Chapter on Gastric Ulcer (page 454).

The introduction of the stomach tube has proved itself as a very useful suggestive measure in several cases of nervous vomiting. Of similar effect is probably also the washing out of the stomach recently recommended by Bendersky.[78] In recent years I have had fairly good success with plain diet, or, perhaps, with synchronous administration of bromides.

Among the medicines there is the most valuable of all, in severe cases, particularly in gastric crises and similar conditions, morphine hypodermically; it is simply indispensable. In less severe cases the application of sedative suppositories may be sufficient, such as

℞ Extracti belladonnæ	0.02
Codeini phosphorici	0.05
Butyri cacaonis	q. s.

ut fiat suppositorium.
Dentur tales doses No. x.
Sig.: One to three suppositories during the attack.

According to Seymour Basch,[79] besides morphine we may consider cerium oxalate (0.1 every two or three hours), antipyrine (0.25 to 1.0 every three or four hours), and strychnine nitrate (0.002 hypodermically). [As mentioned above, the method of using silver nitrate solution (or alumnol) for lavage of the stomach, and followed by bismuth, had an excellent effect in the treatment of gastric crises, and is worth a trial. Whether the silver nitrate had any particular effect on the underlying disease, the tabes, I am not prepared to state in this particular case; but, as the history of the case shows, it has not developed very severely. In the same case cocaine seemed to work beneficially. Hemmeter also prescribes cocaine in cases of this kind.]

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[78] Bendersky, 13th International Congress at Paris; reference, Berliner klinische Wochenschrift, 1900, P. 811.

[79] Seymour Basch, Archiv für Verdauungskrankheiten, 1899, Bd. 5, P. 29.

In other cases of nervous vomiting, menthol in following formula:—

℞ Menthol	2.0
Aquæ destillatæ	120.0
Cognac	30.0

Sig.: One tablespoonful three times a day,

or chloral hydrate (0.5 to 1.0), or bromides (2.0 to 5.0 pro die) have proven successful. In gastric crises, sodium or ammonium bromide combined with sodium iodide (of each 5.0) in water (200.0), one tablespoonful in a cup of milk three times a day, administered during the intervals, seemed to me to have had a beneficial influence upon frequency and intensity of the attacks. The use of the constant current, with the anode in the region of the abdominal plexus [anode within the stomach] and the cathode over the spinal cord, was apparently followed by a palliative influence in several cases. [For the control of vomiting in tabic gastric crises, Hemmeter recommends the following as about the most effective combination he has had experience with:—

℞ Strontii bromati,	
Sodii bromati aa	3.0
Morphini sulphati	0.065
Essentiæ pepsini	200.0

Sig.: One tablespoonful every two hours.

III. Secretory Neuroses of the Stomach.

(1) *Nervous Inacidity, Achylia Gastrica, Apepsia.*

Preliminary Remarks.—As the name achylia (Einhorn) or apepsia (Strauss) indicates, we mean a condition in which the substances, or more correctly proteids, introduced into the stomach are not undergoing any changes, *i.e.*, they pass unchanged into the small intestine. The cause of it is the absence of acid and ferments. On the other hand, as stated by all authorities in unison, the motor function of the stomach is normal or even accelerated (supermotility of the stomach).

The absence of the secretory function may be due to atrophy of the gastric mucosa (anadenia, Ewald; gastric phthisis, G. Meyer), as we have been taught pathologic-anatomically first by Fenwick,[80] and clinically by B. Lewy,[81] Ewald,[82] Jaworski,[83] Rosenheim,[84] G. Meyer,[85] and others. This atrophy may occur as an idiopathic affection, or may complicate other organic gastric diseases, particularly carcinoma (Rosenheim, Hammerschlag). Furthermore, there is—as later writers state—a third form which concerns us here, a form in which essential changes in the gastric mucosa are absent, and which also clinically exhibit the symptom-complex of gastric neurosis.

This standpoint has been taken energetically first by Einhorn,[86] and with some restrictions also by Martius and Lubarsch.[87] Martius considers achylia as some kind of congenital functional weakness, which exposes the stomach to a variety of obnoxious influences, and which may lead to true anatomical changes. It is a fact that the mucous membrane in achylia gastrica is found but rarely entirely normal; *almost always some signs of a more or less pronounced granulating gastritis may be detected.* According to my opinion, therefore, there follows the conclusion that not every case of achylia gastrica finds its anatomical stratum in atrophy of the gastric mucosa, but not that achylia gastrica represents merely a functional disturbance.

The fact stated by Einhorn, that in the course of achylia occasionally normal acid production may be observed (my own experiences confirm this fact), does not speak absolutely against an anatomical process; for the changes need not be in such an advanced state that a total secretory insufficiency follows. Not very rarely we see, even in carcinoma and in neurosis, or physiologically during menstruation, a rapidly alternating disappearance and appearance of hydrochloric acid, a condition which Hemmeter[88] designated very appropriately as heterochylia.

[In his book, "Diseases of the Stomach," Hemmeter gives such an excellent description of all matters pertaining to achylia gastrica, that I consider it a special delight to produce it in the following:—

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[80] Fenwick, On Atrophy of the Stomach and the Nervous Affections of the Digestive Organs, London, 1880.

[81] B. Lewy, Beiträge zur pathologischen Anatomie und Physiologie von Ziegler und Nauwerck, 1886, Bd. 1, P. 201.

[82] Ewald, Berliner klinische Wochenschrift, 1886, No. 32; 1892, No. 26 and 27; Klinik der Verdauungskrankheiten II, 3 Auflage, 1893.

[83] Jaworski, Münchener medic. Wochenschrift, 1888, No. 48 and 49.

[84] Rosenheim, Berliner klinische Wochenschrift, 1888, No. 51 and 52.

[85] G. Meyer, Zeitschrift für klinische Medicin, 1889, Bd. 16, P. 366.

[86] Einhorn, Medical Record, June 11, 1892; Archiv für Verdauungskrankheiten, 1896, Bd. 1, P. 158; Diseases of the Stomach, New York.

[87] Martius and Lubarsch, Achylia gastrica, Leipzig and Wien, 1897.

[88] Hemmeter, Diseases of the Stomach, Philadelphia, 1902, third edition, P. 866.

"*Nature and Concept.*—The term *achylia gastrica* means, literally, without gastric chyle, and was first proposed by Einhorn[89] to designate a class of diseases in which no gastric juice is secreted.

"The affection is found to exist in two varieties—first, the primary, idiopathic, possibly inherited, achylia; secondly, the acquired or secondary achylia. The primary idiopathic or symptomatic achylia is characterised by the fact that absence of secretion is evident before any marked anatomical changes have occurred in the mucosa which could explain the loss of function. It is, therefore, as a rule, not regarded as a result acquired from a real disease, but as an individual peculiarity, possibly an inherited functional debility. There are undoubtedly persons in whom gastric secretion may be absent for years, or permanently wanting; yet who, apparently, may enjoy robust health. The majority of these individuals, however, have suffered from frequent dyspeptic complaints, which are partly of a purely nervous character. In these cases severe anæmic and cachectic conditions are usually absent, and while the general nutrition may occasionally be found disturbed, it is easily remedied with proper dietetic treatment.

"The last-named type of cases demonstrates that the function of the stomach may be permanently lost, so far as its digestive power is concerned, yet with no apparent effects upon the general constitution. A very convincing argument for the compensatory digestive power of the intestine! Lubarsch[90] raises the question whether gastric digestion may not be entirely dispensed with, or whether it is not superfluous, which, of course, implies that the secretion of HCl may possibly be an unnecessary function. There can be no doubt, however, that deficiency of gastric secretion is a disease. Individuals affected with symptomatic achylia are very much more sensitive in general, and more susceptible to gastric diseases, than their fellow-men equipped with normal stomachs. The idea that gastric digestion is superfluous and dispensable impresses us as being a reactive opinion induced by the other extreme view formerly held, according to which the stomach was the most important of all digestive organs. Gastric secretion is by no means a useless function. Lubarsch says: 'Those who have lost it, have one weapon less in the struggle for existence,' and clinical experience teaches that persons who have no secretion of gastric juice are much more liable to diseases of the stomach.

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[89] Einhorn, New York Medical Record, June 11, 1892.

[90] von Martius and Lubarsch, Achylia Gastrica, etc., 1897, P. 74.

When such are attacked by intestinal diseases and this supplementary digestion is interfered with, the prognosis becomes serious.

"Achylia may exist upon a nervous basis, it may be congenital or acquired, in consequence of some organic gastric disease.

"The results of the examination of the gastric contents, in simple uncomplicated achylia, are quite characteristic: the fasting stomach, examined in the morning before any food has been taken, is empty. I have never been able to obtain more than twenty to thirty cubic centimetres of neutral, slightly mucoid liquid; remnants of ingesta of the previous day are never observed. One hour after the Ewald test-breakfast the contents of the stomach have the same appearance as they have in the mouth before they are swallowed. This appearance is claimed by Einhorn and others to be quite characteristic. Contents drawn in this manner are generally slightly acid. Blue litmus paper is very slightly reddened. The total acidity varied in our cases from two to eight. This degree of acidity can be found in the test-meal before it is eaten. Whenever the acidity of the drawn stomach-contents does not exceed that of the meal before it is swallowed, it may be safely assumed that free and combined HCl is absent; in other words, no HCl has been secreted. Whenever the total acidity is equal to four only, it is due to acid that has been introduced in the food; with a total acidity no higher than four, one hour after a test-breakfast, it is, therefore, unnecessary to make further analyses for the detection of HCl. The gastric contents, when filtered and mixed with HCl sufficient to produce the reaction with Congo paper, cannot digest disc of egg-albumen.

"Milk taken by achylic patients may be drawn out twenty or thirty minutes afterward perfectly unchanged, or, rather, uncoagulated. The secretion of pepsine and rennin is, therefore, absent. By proper tests it can also be found that pepsinogen and rennin-zymogen are also wanting. Lubarsch and Martius assert that the isolated loss of HCl, without loss of secretion of pepsine and rennin, does not exist; and for these cases of loss of gastric secretion (not the HCl simply, but all the constituents of gastric juice) the terms anacidity, inacidity, and achlorhydria are not so expressive and logical as the designation 'achylia gastrica.' We may assert, however, on a very large personal experience, that isolated loss of HCl secretion and preservation of formation of ferments does occur. In the progressive destruction of the mucosa accompanying carcinoma and gastritis there are stages in which HCl is totally wanting, and yet, by proper methods, secretion of enzymes, or of the proenzymes, can be detected. All other cases of loss of secretion not due to carcinoma or atrophic gastritis may logically be classed as achylia. A further pronounced sign of achylia is the abnormally small quantity of gastric contents found one hour after the test-breakfast. Biedert,[91] who suffers from this affection himself, found that his stomach was very rapidly emptied, so that he had to draw the contents within forty-five minutes if he wished to obtain any at all.

"Julius Miller[92] found that strong solutions of sodium chloride are very much diluted when they are brought into the human stomach. It is further known that strong solutions of common salt, when brought into the stomach, arrest HCl secretion. The tendency to dilute solutions that are put into the stomach is so persistent that it continues even after the concentration of these solutions has inhibited the HCl secretion. Alcohol, various forms of sugar, dextrine, and peptone are absorbed, and a more or less active excretion of water goes on hand-in-hand and simultaneously with such absorption. In achylia gastrica, however, the stomach differs very much in this respect from the normal organ, since it has then lost its power of diluting the gastric contents.

"The fact that concentrated solutions of sodium chloride inhibit the secretion of HCl has been made available in the treatment of superacidity. From this fact it is very probable that, in achylia, we are dealing not only with loss of the characteristic secretion, the gastric juice with its HCl and ferments, but also that there seems to be no secretion of any kind issuing from the mucosa. The diluting secretion of the stomach is, under normal conditions, not exclusively made up of the normal gastric juice, and we are here confronted with a physiological function of a very complicated character, concerning which very little of a positive nature is known. There is a general consensus of opinion, which we can confirm, that in achylia there is an exceptionally great vulnerability of the mucosa. It is a frequent experience with achylic patients to find that particles of the mucosa showing slight hæmorrhages are unintentionally scraped or torn off during the drawing of the test-meals.

"Lubarsch,[93] Einhorn,[94] Biedert,[95] Cohnheim,[96] and Jaworski[97] have observed this phenomenon, and the first-mentioned author asserts that the vulnerability of the mucosa in achylia is as great as in carcinoma. In achylia it is almost impossible to avoid the scraping off of portions of the superficial mucous membrane, no matter what shaped tube is used, and if it is desired to avoid scraping the mucosa at all, it is safer to use a tube which is entirely closed at its lower end and has but one velvet eye-opening at the side (Tiemann & Co., New York). Scraping off of minute particles is a harmless procedure, but the tearing of larger pieces by suction may be followed by extensive hæmorrhages.

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[91] Biedert, Diätetik u. Kochbuch, etc., 1895.

[92] Julius Miller, Archiv für Verdauungskrankheiten, Bd. 1, P. 233.

[93] Lubarsch, *l.c.*

[94] Einhorn, *l.c.*

[95] Biedert, *l.c.*

[96] Cohnheim, Archiv für Verdauungskrankheiten, Bd. 1, P. 274.

[97] Jaworski, Münch. med. Wochenschrift, 1887, No. 7 and 8.

"Total loss of gastric secretion, even as a consequence of a fully developed atrophy of the mucosa (anadenia), cannot cause anæmia or cachexia *per se*. Those cases in which anæmia has been observed in connection with achylia were most likely complicated by a mechanical insufficency of the stomach, or by other diseases; thus, in some cases, syphilis or tuberculosis, and extension of the atrophic process to the mucosa of the intestine, have complicated the gastric derangement.

"The credit of having first pointed out the association of gastric atrophy with anæmia is usually attributed to S. Fenwick.[98] Both Einhorn[99] and Martius[100] assert that Fenwick's report is the pioneer observation on this subject. As a matter of fact, it was our countryman, Austin Flint,[101] who first called attention to the relation between anæmia and atrophy of the gastric glands. He expressed the opinion that some cases of profound anæmia are dependent upon atrophy of the glands of the stomach. The priority of Flint's publications has been emphasised by Professor William H. Welch.[102]

"Although the anæmia which supervenes in these cases of achylia cannot be directly ascribed to the gastric atrophy, and too much importance was attributed by Flint and others to the state of the gastric mucosa, the reports of this author are, nevertheless, very valuable, because the secondary states, which we have mentioned as really causing the anæmia and cachexia, are most probably brought about by, and owe their origin to, the primary degenerative changes in the gastric mucosa which Flint and Osler have described.

"In the first part of this work I have reported examinations of fragments of mucosa derived from twelve cases of inacidity or subacidity; of these, ten were cases of typical achylia gastrica. In these twelve cases proliferation of glands, with marked round-cell infiltration, was found once. The fragment was apparently normal in two cases, but of the ten cases of typical achylia, granular gastritis and atrophy of the mucosa could be established in nine. In making the diagnosis of simple achylia gastrica we excluded all those cases of permanent loss of secretion evidently due to carcinoma or pronounced chronic atrophic gastritis. In fact, before making these detailed examinations, I supposed it was possible that this form of achylia existed simply as a neurosis, because all of the ten cases which we described occurred in neuropathic patients. I had also inclined to Einhorn's view, that some forms of achylia might be of purely nervous origin. We have since then examined a number of new cases in addition to those reported, making in all fourteen. In none was the mucosa found perfectly normal. It seems improbable that a permanent cessation of a normal function could be caused by a neurasthenic condition. This explanation of achylia would be justifiable only in case we could demonstrate that in this affection the gastric mucosa was perfectly normal. To our knowledge, there is no case of well-authenticated achylia on record in which a cure or an improvement in the neurasthenia was reported to have cured or improved the secretory defect.

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[98] Fenwick, Lecture on Atrophy of the Stomach, The Lancet, July 7, 1877; also on Atrophy of the Stomach and Certain Nervous Affections of the Digestive Organs, London, 1880, J. & A. Churchill.

[99] Einhorn, *l.c.*, P. 321.

[100] Martius, *l.c.*, P. 16.

[101] Austin Flint, The American Medical Times, 1860. The further contributions of Flint to this subject are to be found in the New York Medical Journal for March, 1871, and in his Principles and Practice of Medicine, P. 477, Philadelphia, 1881.)

[102] Welch, System of Medicine by American Authors, Vol. II, P. 616.

"There are, no doubt, highly nervous patients who secrete HCl normally, but under the influence of the nervous excitement and apprehension coincident with the drawing of the test-meal, the gastric secretion is temporarily inhibited. I have seen three such cases in which I could not detect HCl at six consecutive analyses, but a normal secretion was found in the vomited matter brought up by the patient at home. Later, the secretion was also found normal in the test-meals drawn at my office. The question has also suggested itself, whether achylia gastrica could not be the cause of the neurasthenia. Nor on this point are there any authenticated observations. Experience has taught, however, that neurasthenic disturbances disappear in these cases with an improvement of the general condition, while the achylia continues, which would not be the case were the latter the cause of the neurasthenia. The loss of function in this disease is not a relative or transient one, but it is absolute and permanent. Biedert[103] gives it as his opinion that the persistent loss of HCl and ferments gives the impression of a lasting defect, not of a variable increasing or decreasing inhibition. The absence of the gastric secretion is the same, whether the patients are very much run down and emaciated and subject to much suffering or whether they are in a state of good health. The supposition of Biedert, that there may be a great many who possess this defect and are unaware of it, has been verified by a number of observations among the students at my clinic. While studying the question whether the normal healthy stomach contained digestiv juice in the fasting condition, we discovered an athletic, robust studen who had no HCl whatever in his stomach, whether fasting or after meal The total acidity, taken after test-meals on six different occasions, varie between one and four; as these analyses were made shortly before th examinations for the degree of M.D., we did not inform the candidate o the physiological defect in his stomach, fearing that it might cause hi some mental annoyance, and for all that we know he may still be unawar of his achylia and continue in vigourous health. My results concerning th condition of the gastric mucous membrane are in accordance with thos of Cohnheim,[104] Einhorn,[105] Hayem,[106] and have recently been supporte by Martius and Lubarsch,[107] and H. Strauss.[108] The results of the ver exact investigations of these last authors make it probable that a mor or less pronounced granular gastritis exists in the majority of cases o achylia.

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[103] Biedert, *l.c.*, P. 173.

"The anatomical changes, however, are not, in all cases, sufficientl advanced to explain the permanent loss of function. There is no indication at present for determining whether glandular gastritis is the caus or result of achylia. It is self-evident that a weak gastric parenchym should be less resistant to exterior detrimental influences—such as bacterial invasion—than a robust gastric tissue. The secretion of HCl bein the normal disinfectant, though not an absolute antiseptic, it largely protects the mucosa from infection.

"It is evident from what has been said in the ætiology of the variou diseases of the stomach that the organ is exposed to many external aggressions of a thermic, chemical, mechanical, and bacterial nature, and it i a matter of astonishment what intense maltreatment a healthy stomac will endure without reacting pathologically. It is, therefore, conceivabl that the anatomical loss of the glandular apparatus will render thos individuals afflicted with primary simple achylia more susceptible t bacterial invasion. Most observers agree that the increased vulnerabilit of the mucosa goes hand-in-hand with the loss of secretion; this lessen the power of resistance, and eventually induces a state of chronic granular gastritis, effected by causes which a healthy stomach would resis without any change.

"*Symptoms.*—The disturbances of function may long remain latent Persons with achylia may for many years have no subjective or objectiv disturbances of any kind; but sooner or later dyspeptic complaints arise. The subjective sensations are not characteristic, but are essentially those of nervous dyspepsia, accompanied by eructation, fulness, and pressure after eating, gradually leading to attacks of severe gastralgia. The symptomatology, as based upon the complaints of the patient, is most accentuated in neurasthenics. In persons with a perfectly sound nervous system achylia may exist, and the individual may be unaware of it; this is proved by the case of the medical student reported in the preceding. Oppler[109] has reported a number of cases which make it probable that loss of gastric secretion predisposes to diarrhœa and intestinal catarrhs, which are not benefited until the achylia is discovered, when rational treatment effects improvement. The personal description which Professor Biedert[110] gives of his own case is a weighty argument pointing to the fact that achylic patients are very much predisposed to diarrhœa. Among the achylic patients which I have studied (fourteen in all), I observed attacks of diarrhœa in five. So far as I could determine, the colon and the duodenum were in normal condition. I also studied the state of the duodenum by my method of duodenal intubation, showing the pancreatic and hepatic secretions to be normal. This makes it probable that these diarrhœas are possibly not due to an extension of the anatomical changes in the stomach to the intestine, but to fermentative processes, developed in the absence of HCl secretion. These diarrhœas confirm Bunge's view that at least one effect of the HCl secretion is that of a partial antiseptic.

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[104] Cohnheim, *l.c.*

[105] Einhorn, New York Medical Record, June 27, 1896.

[106] Hayem, Allgem. Wiener med. Zietung, 1894, No. 2-17.

[107] Martius and Lubarsch, Achylia Gastrica, P. 112-170.

[108] H. Strauss, Virchow's Archiv, Bd. 154.

"Martius' conclusions[111] are the following: achylia gastrica is due to two conditions: (1) a primary secretory debility of the stomach, constituting simple achylia gastrica; (2) atrophy of the gastric mucosa (anadenia), which is secondary achylia gastrica. The primary achylia gastrica is either congenital or developed on the basis of a very early predisposition. It is associated with inherited debility of the nervous system, and prevails among so-called neuropathic patients.

"Primary secretory debility of the stomach is an individual peculiarity, which may remain latent for years, and without demonstrable detriment to the general organism. This is particularly the case when the motor function is well preserved, and the motor, secretory, and resorptive functions of the intestine continue normal.

"The mucosa, which is devoid of secretion, exhibits a diminished vital resistance to all external detrimental influences. This explains the fact that anatomical alterations of varying intensity are rarely absent in simple achylia gastrica. The structural changes bear no proportion relation to the absolute gravity of the loss of function.

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[109] Oppler, Deutsche med. Wochenschrift, 1896, No. 32, S. 511.

[110] Biedert, *l.c.*

[111] Martius, *l.c.*, P. 101.

"It is, therefore, probable that there are forms of so-called atro of the gastric mucosa (the primary non-carcinomatous anadenia) wh develop preferably on the basis of this congenital secretory weakness the stomach.

"Accordingly, there are gradual transitions, clinically and anaton ally, from congenital simple achylia with but immaterial alterations the mucosa, to achylia with chronic granular gastritis eventuating in c plete atrophy of the secretory mucosa.

"The grave results for the total organism (progressive anæmia, n nutrition, etc.), which have been ascribed to the latter type, do not reality develop until the mucous membrane of the intestine is extensiv involved by the atrophy.

"*Pathological Histology.*—The investigations made by the auth quoted in the literature show, in general, a marked increase in the int stitial connective tissue. The surface epithelium contains many gob cells. The vestibules to the glandular alveoli are very tortuous, and dilated that they resemble minute cysts, filled with homogeneous, sligh granular masses, that stain with acid aniline. The epithelial cells lin the vestibules present marked variations in structure and staining qua ties. Those most prominent are: (1) ordinary, long, cylindrical epithel cells; (2) somewhat shorter, cylindrical cells with dark protoplasm a dark staining nucleus, the upper end of which has disappeared; (3) gob cells; (4) cells as in type 2, but with a very dark protoplasm (Stöhr cell (5) cells with a marked fuchsinophilic granulation. In some vestibu only cells answering to the description of type 2 are found, and in th an abundance of mitotic figures. In other vestibules we find goblet c in addition to these. There are very few vestibules which contain norn surface epithelium.

"Among the other characteristics that were found in freshly harder stomachs of achylic patients are: (1) immigration and permeation leucocytes; (2) the occurrence of mitoses in the surface epithelium a in that lining the vestibular alveoli. The author's cases were especia examined with regard to atypical or pathological mitoses, but the resu were negative. (3) occurrence of acidophilic leucocytes; (4) frequency goblet cells; (5) occurrence of so-called Stöhr's and Nussbaum's cells; occurrence of hyaline spheres.

"Referring to No. 1 of the above observations, it should be sta that Sachs[112] has found an abundance of lymph-cells migrating throu the surface epithelium and glandular substance. The pyloric region seems to be more invaded than any other part of the stomach. Stintzing considers the immigration of leucocytes in the normal stomach a very rare occurrence. Permeation of the gastric mucosa with leucocytes at the height of digestion is a normal occurrence, and has been frequently observed in animals. The difference in the achylic stomach, with regard to the permeation of leucocytes, is simply one of degree. Lubarsch found that the glandular lumina were actually packed full with acidophilic leucocytes; this property has not been found in the leucocytes of the normal stomach. It is probable that the invasion of the mucosa with acidophilic leucocytes to such a degree as Lubarsch describes is pathological.

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[112] Sachs, Zur Kenntniss d. Magendrüsen b. krankhaften Züstanden, Breslau, 18

"Concerning No. 2, the presence of mitoses in the epithelia of the normal stomach is denied by Sachs[113] and Oppel,[114] and the occurrence of karyokinetic figures in the chief and border cells is extremely rare. For a closer study of the character and significance of the mitotic processes I refer to my article.[115] The hyaline spheres which Lubarsch describes are composed of cell granules that have become confluent and enlarged, but are still contained within the body of the original cell. These hyaline spheres are considered pathognomonic for atrophic processes in the gastric mucosa.

"The histological changes found by various authors in achylia, and which we have been enabled to confirm in cases which we had opportunity to examine at autopsies shortly after death, indicate the proliferation of the interstitial connective tissue, the occurrence of acidophilic migrating cells; and, in addition, the disappearance of the specific glandular elements and cell proliferation emanating from the vestibules of the glands; also transformation of the gastric mucosa into intestinal mucosa. The process eventuates in complete atrophy of the mucosa. Einhorn has reported a case of achylia in which a bit of gastric mucosa was found in the wash-water, which under the microscope appeared normal. We obtained normal mucosa from two cases of achylia, when strips were cut from achylic stomachs running from the œsophagus along the greater curvature to the duodenum; on serial sections made at intervals of one inch apart, small areas of microscopically normal mucosa were found, particularly near the cardia, while most other portions of the stomach showed distinct atrophic changes, with profuse immigration of leucocytes, and proliferation of the interstitial connective tissues. A small bit of mucosa accidentally found in the wash-water does not indicate the state of the entire stomach. When such normal fragments are found in achylia it is still probable that other portions of the stomach may be diseased, and what may be a normal condition in a fragment from the fundus or pyloric region, will be pathological for the intermediate zone.[116] We do not, therefore, consider the evidence satisfactory that achylia may exist with a perfectly normal gastric histology.

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[113] Sachs, *l.c.*

[114] Oppel, Lehrbuch der vergleich. mikroskop. Anatomie d. Wirbelthiere, Bd. 1, Magen.

[115] Hemmeter, Histological Studies Relating to the Early Diagnosis of Cancer of the Stomach, Philadelphia Medical Journal, February, 1900.

"In the great majority of cases of achylia, a progressive atrophic gastritis may be found to exist. There may be periods in the history of achylia when this condition exists without any apparent alteration in the gastric mucous membrane, and the fact that most patients do not consult the physician until the process has developed to a very advanced state may explain the observation that the occurrence of achylia with perfectly normal stomachs is thus far supported by very few reliable microscopic examinations of gastric tissue fragments. All achylic patients give a history of years of gastric disturbances when they first present themselves for treatment, the anamnesis thus making it probable that the gastric changes must have progressed very far. In the case of the healthy medical student in whom we found achylia on six different examinations, we did not succeed in obtaining a piece of the gastric mucosa. In these cases frequent examinations for fragments of mucosa are necessary to decide the relation between the histological alteration and the clinical history. These examinations should be made at frequent and regular intervals, and in case of autopsies on achylic patients the stomach should be previously preserved by pouring in alcohol or Zenker's fluid within a half hour after death, so as to prevent autodigestion. What relation exists between the atrophic process of the intestines and that of the stomach is unknown. It may be a direct continuation of the progressive gastritis, since it is very probable that the same detrimental agencies that cause the disease of the stomach give rise to the intestinal atrophy. One might assume also that excessive demands are made upon the digestive power of the intestines in the absence of the preparatory digestive function of the stomach. Again, it is probable that bacterial fermentations occur to a much greater degree when the disinfecting power of the HCl is lost. In two autopsies on subjects who had shown the symptoms of achylia, the author observed that the celiac axis, and all branches arising from it, were of unusually small size. The gastric arteries were smaller than those of normal stomachs. The intestinal and mesenteric arteries were also smaller in diameter than normal. The dimensions of the hepatic and splenic arteries were smaller. The arteries of the heart, spleen, kidneys, and liver appeared normal in size. On injecting the arteries of the stomach from the celiac axis, the diminutive calibre of the arteries was evident even without micrometric measurements. There was, in this case, no atrophic gastritis.

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[116] Martius and Lubarsch, *l.c.;* also Hemmeter, *l.c.*

"*Ætiology.*—Aside from the probability that achylia may be either congenital or developed upon a neuropathic basis, not much is known of the causation of the progressive atrophic gastritis. It has been supposed that bacterial infection is an ætiological factor in bringing about this state of the mucosa. We may conceive the bacterial invasion to have occurred in a similar manner to that pictured under the head of ulcus carcinomatosum. It is not known whether this bacterial invasion is a cause or result of these processes. Syphilis and tuberculosis may be predisposing factors."]

Differential Diagnosis.

Rarely, or not at all, there will be a reason for mistaking the condition for gastric ulcer, stenosis of the pylorus, or primary stasis insufficiency, if the results of the examination of the gastric contents are taken into consideration.

But the distinction between achylia gastrica and gastritis chronica inacida, as well as between achylia and carcinoma, may be very difficult.

As already mentioned, there is no possibility of a distinction between achylia gastrica of a nervous or an organic nature. Even the examination of particles of the mucous membrane, which formerly I considered of great value, cannot, according to the investigations of Lubarsch[117] and Leuk,[118] be regarded any longer as decisive, because they do not give us a complete picture of the anatomical condition of the *entire* gastric mucous membrane. Only the finding of mucus, epithelial cells, and leucocytes, which point to a productive-inflammatory process of the gastric mucosa, may be decisive to a certain degree. As a matter of course, the reverse does not hold good.

Sometimes the distinction between achylia gastrica and carcinoma may be very difficult. Of course, the fact emphasised by Martius, Einhorn, and others, that in achylia gastrica, in contrast to carcinoma, motor disturbances are absent, is of importance; but there are also cancers of the lesser curvature, and indeed even of the pylorus (as I observed in a case), in which every trace of motor disturbance may be missing for a long time.

In such cases solely the clinical course is decisive, especially the

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[117] Lubarsch, Martius and Lubarsch, *l.c.*

[118] Leuk, Zeitschrift für klinische Medicin, 1899, Bd. 37, P. 296.

favourable reaction of the patients upon increased feeding, that is,
permanent increase of weight and strength. The fact also that t
patients had complained of slight digestive troubles for long years rath
speaks against a malignant affection. Moreover, conditions of re
cachexia are seldom observed in patients suffering from achylia, althou
loss of weight (usually of artificial nature) is a usual occurrence.
greater importance, appears to me, the absence of regularly occurring, s
called occult bleedings, which, at least to the present time, I never cou
find in achylia gastrica, whereas in cases of gastric cancer they are ve
frequent. There occurred, however, a few cases which, in spite of se
eral weeks' observation and in spite of consideration of the above-me
tioned factors, were and remained doubtful.

Treatment.

In the treatment of achylia gastrica the main task must be the reli
of the stomach from its work through appropriate preparation of t
food—in one word, through predigestion. At the same time, anoth
requirement is complied with, namely, relieving the intestines, whic
under these circumstances, are burdened with a far larger task. Furthe
more, diet must be of such a condition that an *increase* of weight follow
and by no means a reduction of weight.

But with this, as Martius emphasises in his very valuable "therapeut
notes" to achylia gastrica, the restrictions ought to stop. A modificati
of the diet—too extended and, as I may say, affected—according
grammes and cubic centimetres, is, as for dyspeptics in general, n
only entirely dispensable for patients suffering from achylia, but rath
injurious. Patients in whom each food is controlled and measured
square and compass become finally "willing slaves to their gastropho
imaginations" (Martius).

Only in the presence of certain complications (diarrhœa, constipatio
accurate directions for diet corresponding to the circumstances should
given, but it must always be the physician's task to train the patients
become again persons who enjoy the joys of life, and to become ind
pendent from too narrow a dietetic limitation. When neurasthen
symptoms require it, appropriate curative measures, such as hydrotherap
physical culture, electrotherapy, ought to be called on for assistance.

Medicinal treatment is but secondary to the just-mentioned measur
It is doubtful whether any effect can be attained through the administr
tion of smaller or larger doses of hydrochloric acid, especially as vari
tions occur in the course of achylia as well as in that of gastritis. A

objective amelioration, that is, a visible improved chymification through the administration of hydrochloric acid, has, according to numerous observations of authors, not been obtained.

Whether better results will be obtained through the administration of the new preparation "gastérine" (see pages 359 and 425), is still uncertain. My own experience is still too small.

[As far as a few cases under my observation indicate, the natural gastric juice (gastérine) is certainly worth a trial, as it acts entirely differently from the commercial hydrochloric acid. In cases of achylia gastrica I have used caroid with greatest advantage to the patient. In cases of this kind it is particularly the pancreas powder which may be of greatest advantage; the same holds good of diastase. While hydrochloric acid is of but slight value as a true digestive in these cases, I nevertheless consider it valuable as a stimulant for the gastric peristalsis, especially when administered in doses of about 30 to 60 drops about half an hour to an hour before meals.]

(2) *Hyperchlorhydria.*

(Superacidity. Hyperacidity).

By hyperchlorhydria (or superacidity) we understand a condition of abnormal glandular irritation which is manifested through an increase of normal hydrochloric acid secretion, and also of ferment production (total hydrochloric acidity after a test-breakfast amounts to more than 2.5 per mille). Superacidity by itself occurs in a great variety of gastric affections, and even, as Verhaegen[119] has shown, in persons with a *healthy stomach.* A part of the clinical picture of hyperchlorhydria must be accordingly the increased irritation of the gastric mucous membrane due to the influence of the acidity. As we will see, the irritation is not associated with definite limits of acidity, and may occur although the amount of acidity is relatively low. The deciding point is given accordingly by the clinical symptom-complex.

Is there at all a superacidity as a functional disturbance? This question must be undoubtedly affirmed, and was also confirmed several years ago by Oesterreich,[120] who reported a case of superacidity, in which at the autopsy no pathologic changes at all were found aside from numerous erosions. The clinical history described by v. Leyden[121] shows very instructively the type of a gastric neurosis, and that investigator sta he has seen a large number of similar cases.

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[119] Verhaegen. Cellule T. xiv f. 1; quoted in Centralblatt für klinische Medicin, 1899, P. 22.

[120] Oesterreich, Deutsche medicinische Wochenschrift, 1895; Vereinsbeilage, No. 21.

[121] v. Leyden, *ibid.*

The occurrence of a purely *neurogenous* superacidity can accordin not be doubted any longer. More difficulties are found in complicat cases. In these there may have existed first a real and pure superacidi which may have been followed later on by erosions, ulcer, spastic organic stenosis of the pylorus, atony, etc. The originally nervous ch acter of the affections becomes lost under cover of the entirely chang symptom-complex.

Diagnosis.

(*a*) *Subjective Symptoms.*—The subjective signs consist of pyro (heartburn), eructation of sour-tasting substances, burning and bori sensation in the epigastrium. Heartburn occurs particularly after ing tion of sour, salted, or greasy substances, but is not present, or at lea only to a slighter degree, after ingestion of meat. The sensation of hea burn (pyrosis hydrochlorica) is probably caused through irritation of t œsophageal nerves by the ascending acid substances.

The appetite is usually very good. Frequently there is bulimia ass ciated with gnawing pain in the epigastrium, which is very soon reliev by alkalies or milk, egg-albumen, meat, crushed almonds. The bowels a usually very much constipated; in some rare cases, however, there may diarrhœa.

(*b*) *Objective Symptoms.*—These may be divided into physical a chemical signs. As to the former, it must be stated that in pure cas any changes of the organ (tenderness on pressure, distension, dilatatio may be entirely absent. In not entirely pure cases gastric dilatation frequently found, which, on careful examination, manifests itself mechanic insufficiency (myasthenia). It needs a thorough considerati to explain in each single case how far the overburdening of the stoma must be considered as primary, and the superacidity as secondary. T chemical examination of the stomach-contents after the test-breakfa shows degrees above the normal one. On the other hand, I frequent made the observation that symptoms of superacidity may be present *spite of perfectly normal acid secretion.* Schüler[122] recently confirmed t same fact, and designates such a superacidity as "hyperaciditas [supe aciditas] larvata. [Cases of this kind are, according to my experienc not at all infrequent. I had under my observation a number of patien suffering from all the subjective symptoms of hyperchlorhydria, althoug the amount of hydrochloric acid on many examinations, at various times and after various test-meals, never amounted to over 0.2 per cent.; in several cases of this type there was, however, one abnormal condition, namely, a disturbance in the digestion of amylum, and this in spite of the normal amount of hydrochloric acid.] In such cases, as Talma[123] suggests, a particular *hyperæsthesia of the nerves of the stomach* towards acids must be assumed.

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[122] Schüler, Deutsche medicinische Wochenschrift, 1900, No. 19.

For several years I have had under observation a man in his thirties, who complains of severe occasionally occurring burning in the epigastrium, which is promptly relieved by large doses of sodium bicarbonate. The epigastrium itself is tender on pressure, stomach not dilated. Repeated examinations of the stomach-contents exhibit HCl values varying between 1.5 and 1.8 per mille. A brother of the patient suffers from the same gastric affection.

The superacidity may also be demonstrated according to Riegel's suggestion, indirectly from comparison of the amylum and meat digestion, whereby Riegel's test-meal is very appropriate. If the stomach-contents are removed after three or four hours and filtered, we find in the remnants on the filter large quantities of amylum and complete absence of meat residues.

According to Schüler, the specific gravity of the gastric contents in superacidity is diminished, compared to that in subacidity; it is usually below 1016 (normal is 1015 to 1020). The diminution must be referred to diluting the secretion of the stomach.

Among the objective symptoms the condition of the *urine* is also to be taken in consideration. The urine is distinguished by decrease of acidity or alkaline reaction, and simultaneous decrease of the chlorides. On the other hand, the phosphates are stated to increase; I frequently observed phosphaturia in patients with superacidity. But as the reaction of the urine, as well as the amounts of chlorides and phosphates, do not depend only upon the gastric digestion, numerous variations may evidently occur. A decisive diagnostic importance can certainly not be attributed to the condition of the urine.

Differential Diagnosis.

Differential-diagnostically, we must consider ulcus ventriculi, gastritis acida, myasthenia, gastrochylorrhœa.

The differential diagnosis between hyperchlorhydria of nervous origin and chronic ulcer (without previous hæmorrhage) is facilitated through the following factors: in cases of ulcer circumscribed pain on pressure is found, while in nervous superacidity it is usually lacking; likewise dorsal algetic points, mentioned in the Chapter on Ulcus Ventriculi (p 432) are absent, though they may occasionally be missing in ulcer. In ul the intensity of pain, at least in typical cases, stands in direct proport to the quality of the ingesta; in superacidity such a connection cannot observed. In spite of these differential factors, the differentiat between nervous superacidity and gastric ulcer frequently meets di culties, because occasionally superacidity may follow an ulcer or may p cede it. In the Chapter on Ulcus (page 434) we mentioned the numer deviations from the classical symptoms of ulcer. In cases of this kind experiment of a test ulcer cure would prove useful; its favoura result would, though not with certainty, speak for a probable ulcer.

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[123] Talma, Zeitschrift für klin. Medicin, 1884, Bd. 8, P. 407.

The differentiation of nervous superacidity from gastritis acida easy in some cases; in other cases it is difficult or even impossible. T diagnosis is readily made, when in the fasting stomach and after te breakfast much mucus is constantly found, besides superacidity. Impo ant also is the result of microscopic examination of the gastric mu (page 410). The examination meets difficulties when the said criteria a missing; according to my experience, gastritis may exist frequently a complication with ulcus ventriculi; according to my opinion, sup acidity occurring not infrequently in an ectasia may be a manifestation beginning gastritis; of course, this fact is not yet proven. In some ca the same may perhaps happen through examination of small muc particles.

The distinction between mechanic insufficiency and nervous acid may encounter difficulties, since the subjective symptoms are congru in many cases and increase of acidity is frequently observed in myasthen As, however, the purely nervous superacidity occurs without motor insu ciency, we had best, as mentioned above, if insufficiency is present, rega the increase of secretion as secondary. The same is to be said in cases gastroptosis. The said anomalies may also be associated with the clini picture of general neurasthenia or nervous dyspepsia, and it becomes oft very difficult to decide between cause and effect.

As to differentiation between superacidity and chronic continuo gastrochylorrhœa, see page 536.

Treatment.

The treatment of superacidity has first of all the function to avo abnormal stimulation of the glands by an appropriate diet. Which su stances stimulate the glands? It has been decided, through the expe ments of Pawlow[124] and his disciples on dogs with œsophageal and gastric fistulæ, that meat and bouillon invite more abundant and longer lasting juice secretion than, for instance, milk and bread.

Numerous examinations on human beings by v. Jaksch,[125] v. Sohlern,[126] Jürgensen[127] and Justesen,[128] Hemmeter,[129] Backmann,[130] Arth. Meyer,[131] Max Buch,[132] and others have yielded the same results.

On the other hand, investigations made on dogs by Lobassow,[133] and Wolkowitsch,[134] and on human beings by Ewald and Boas,[135] Akimow-Peretz,[136] Strauss and v. Aldor,[137] Backmann,[138] and Buch[139] have shown that the ingestion of fat decreases the secretion of acid.

These investigations, the interesting details of which cannot be discussed here, caused a certain change in the treatment of superacidity. After v. Sohlern, already supported by his experiments, had recommended food rich in carbohydrates, Jürgensen,[140] of Copenhagen, spoke zealously in several articles in favour of a change of dietetic principles which hitherto had been recognised as valid in the treatment of superacidity. Several writers, among them Hemmeter, Backmann, and Buch, followed him essentially, while, on the other hand, disagreeing views (Schüle, Riegel, Arth. Meyer, and others) were not missing.

The real fact in these undoubtedly meritorious investigations is that

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[124] Pawlow, Die Arbeit der Verdauungsdrüsen, Wiesbaden, 1898. [English edition: The Work of the Digestive Glands, by Dr. W. H. Thompson, London and Philadelphia, 1902, P. 93 *et seq.*]

[125] v. Jaksch, Zeitschrift für klin. Medicin, 1890, Bd. 17, P. 383.

[126] v. Sohlern, Berliner klin. Wochenschrift, 1891, No. 20 and 21.

[127] Jürgensen, Archiv für Verdauungskrankheiten, 1897, Bd. 3, P. 225.

[128] Jürgensen and Justesen, Zeitschrift für diätetische und physikalische Therapie, 1900, Bd. 3, P. 541.

[129] Hemmeter, Archiv für Verdauungskrankheiten, 1898, Bd. 4, P. 23.

[130] Backmann, *ibid.*, 1899, Bd 5, P. 336.

[131] Arth. Meyer, *ibid.*, 1900, Bd. 6, P. 299.

[132] Max Buch, Zeitschrift für diätetische und physikalische Therapie, 1901, Bd. 4, Heft 3 and 4, P. 189 *et seq.*, and 330 *et seq.*

[133] Lobassow, ref. Archiv für Verdauungskrankheiten, Bd. 2, P. 499.

[134] Wolkowitsch, refer. *ibid.*, Bd. 4, P. 380.

[135] Ewald and Boas, Virchow's Archiv, 1886, Bd. 104, P. 271.

[136] Akimow-Peretz, ref. Archiv für Verdauungskrankheiten, 1898, Bd. 4, 397.

[137] Strauss and v. Aldor, Zeitschrift für diät. und physik. Therapie, 1898, Bd. 1, P. 117.

[138] Backmann, Zeitschrift f. klin. Medicin, 1900, Bd. 40, P. 224.

[139] Max Buch, *l.c.*

[140] Jürgensen, *l.c.*

carbohydrates and fats* are usually tolerated much better than was fo
merly assumed. It must, however, be stated that numerous individu
variations happen to exist. Every experienced physician will rememb
patients in whom very sweet cakes or pastries always caused severe hea
burn; ingestion of fats also is by no means well borne by all patien
suffering from superacidity.

The term "carbohydrate diet" needs a more accurate definition, or rather
striction. If you number among these carbohydrates salads, raw fruit, vegeta
roots, I doubt very much whether great results can be obtained by means of such
carbohydrate diet. The advocates of the new method probably think only of ri
farinaceous dishes, purées of potatoes and vegetables, whereby the method of pre
ration and the physical condition play an essential part.

But if we concede a favourable influence to a nourishment consisti
entirely or mainly of carbohydrates or fats, we must strictly distingui
between the influence of the same upon the single act of digestion and th
upon the glandular secretion in general.

As to the former, it may, as already mentioned, be accepted as po
tive that carbohydrates and fats in appropriate preparation are usua
well tolerated and well assimilated by patients suffering from superacidi
I could, however, not convince myself that the subjective troubles disa
pear less quickly though meat diet has been allowed in normal limits
the same time. Closely associated with this question is the fact discover
by Backmann,[141] *that raw meat, meat-juice, and solutions of Liebi
beef tea strongly stimulate the secretion of gastric juice, but that cook
meat becomes the more inactive the longer it has been exposed to cookin
and that finally it entirely loses the effect upon gastric secretio*
Accordingly patients suffering from superacidity would be harmed by ra
or rare meat, but not by meat well done.

As to the real curative effect of the carbohydrate-fat diet, the
exist, as far as I know, no convincing proofs for the permanent decrea
of the secretion of acidity. It is true, Akimow-Peretz and Buch describ
similar experiments, but, as far as I can learn from the report of the sa
experiments, the decrease of the hydrochloric acid was not considerab
and increased anew after the fat diet was stopped. In one case Bu
observed a decrease of hydrochloric acid from 122 to 70 or 75, but unfo
tunately the patient received soda besides fat. Experiments made

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* The administration of fat in superacidity is not entirely new. v. Noord
has already proved the excellent consumption of fats in superacidity; I myself e
phasised already in the second edition of this work [1895, p. 248], that fats in go
substance are very well tolerated in superacidity.

[141] Backmann, Experimentelle Studie, Akademische Abhandlung, Helsingfors, 18
quoted after Buch.

my suggestion by Arth. Meyer[142] showed that the hydrochloric acid curves do not vary essentially with long-continued purely carbohydrate and animal diet; on the contrary, they slightly increase when the food is poor in proteids.

The present results of investigations are, as readily seen, still too full of contradictions to induce us to abandon our present principles at this time. The best food in superacidity remains, as heretofore, the mixed diet, whereby fats and carbohydrates need not be feared, but are only to be administered in suitable forms. But all sour, greasy, piquant, and too sweet substances ought to be banished from the table for patients of this kind.

An important dietetic rule in superacidity is that of never allowing the stomach to become entirely empty, because a frustrate secretion of gastric juice and with it pain and burning are provoked. Alcohol in form of a well-brewed beer and of wine (especially the red Rhine wines or the "stomach-friendly" Voeslau) may be allowed in moderate quantities, but Moselle wine, cider, champagne are to be forbidden.

A great part in the treatment of superacidity is played by the so-called *acidulous waters* (*Säuerlinge*), which act as a sedative partly through the carbonic acid, partly neutralise the superabundance of acid through the alkali carbonates. The most frequently used acidulous waters (their amount of CO_2 and Na_2CO has been described in Part I, page 318) are Bilin Sauerbrunnen, Neuenahr Sprudel, Krondorfer, Fachingen, Giesshübler, Apollinaris, Selters, etc. The French especially recommend the water of Vals, Contrexéville, and Vichy [Saratoga Vichy, rich in CO_2, Londonderry Lithia]. The said acidulous waters may be taken alone, or may be administered with an addition of wine, milk, etc. Pastilles are prepared of some of the acidulous waters (for instance, Bilin and Vichy), and serve readily for portable purposes.

The medicines in their stricter sense aim partly at symptomatic effect, partly at direct cure. To these belong the much used and abused alkalies, above all the sodium bicarbonate, sodium citrate, ammonia-magnesium phosphate, magnesia usta, calcium salts, Carlsbad salts, and other preparations. I prefer the fixed earthy alkalies, because their administration does not cause abnormal carbonic acid formation, which occasionally becomes very troublesome to the patients. Moreover, the magnesium salts are preferable because they have a mild laxative action. As atropine (see below) is supposed to have an inhibitory effect upon glandular secretion, a combination of the said salts with small doses of extract of belladonna is to be recommended, according to the following formula:—

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[142] Arth. Meyer, *l.c.*

℞ Magnesiæ ustæ	15.0
Extracti belladonnæ	0.1

M. f. pulvis.
Sig.: One-fourth to one teaspoonful three times a day.

The carbonated sodium and magnesia water recommended by Jaworski, which are mentioned in the Chapter on Gastrochylorrhœa, are, of course, indicated also in superacidity. As to the effect of alkalies upon gastric secretion, the most important points have been discussed in the General Part (page 363).

Apparently very rational is the administration of atropine, which first Riegel,[143] then Arth. Schiff[144] and v. Aldor,[145] have shown that it greatly diminishes the secretion of hydrochloric acid and pepsine, though, according to Arth. Schiff, the pepsine secretion is not influenced. But the experiences so far known, particularly those of v. Aldor, give the practical administration of atropine but a limited place in our armamentarium against superacidity. My own numerous investigations did not yield a distinct and regular effect upon the subjective troubles and upon the decrease of the acid secretion.

More favourable, perhaps, in such cases is the application of concentrated solutions of sugar, which, according to investigations by Verhaegen,[146] Riegel,[147] Strauss,[148] and v. Aldor,[149] cause a decrease of acid production by transudation. According to v. Aldor, the most preferable sugar is the fruit-sugar, particularly as it possesses the quality of binding the greatest amount of hydrochloric acid.

When stubborn constipation exists, Carlsbad salt is most useful; in other cases dietetic measures combined with the necessary energy of the patient are almost always sufficient.

Among the mineral waters proper, the thermal waters of Carlsbad, Vichy, and Neuenahr are to be considered. They act partly as sedative for the nerve-fibres supplying the glands, partly neutralising the acids.

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[143] Riegel, Zeitschrift für klinische Medicin, 1899, Bd. 37, P. 381.

[144] Arth. Schiff, Archiv für Verdauungskrankheiten, 1900, Bd. 6, P. 187.

[145] v. Aldor, Zeitschrift für klinische Medicin, 1900, Bd. 40, P. 248.

[146] Verhaegen, Cellule, T. xii, Fasc., quoted in Centralblatt für klinische Medicin, 1897.

[147] Riegel, Die Erkrankungen des Magens, 1877, P. 718.

[148] Strauss, Zeitschrift für klinische Medicin, 1900, Bd. 29, P. 248.

[149] v. Aldor, *l.c.*

On account of frequent synchronous constipation, the Carlsbad thermal waters are to be made more efficient by addition of appropriate quantities of Carlsbad salt.

(3) *Periodical Flow of Gastric Juice* (*Reichmann*), *Gastroxynsis* (*Rossbach*[150]).

Preliminary Remarks.—By periodic flow of gastric juice we understand a disease in which paroxysms of grave dyspeptic symptoms occur in association with severe cramp-like pains and vomiting of larger or smaller quantities of acid gastric juice. With the onset of the vomiting relief may appear temporarily or permanently, though the paroxysm may last for several days. The type of periodic flow of gastric juice, in which before or at the beginning of the attack severe boring headaches occur, has been described by Rossbach as a special neurosis and named *gastroxynsis*. There is, however, no doubt that this type represents a special variety of the "periodic flow of gastric juice" and cannot claim a separate position.

The periodic flow of gastric juice may occur either as an idiopathic secretory neurosis or as a reflex neurosis induced by affections of the central nervous system (tabes, myelitis, progressive paralysis); particularly the vomiting of gastric juice observed during gastric crises in patients suffering from tabes has often been claimed to represent a form of periodic flow of gastric juice. The vomiting of acid gastric juice is, however, according to the investigations of v. Noorden,[151] Boas,[152] Bouveret,[153] and Seymour Basch,[154] by no means a constant symptom in gastric crises; in some cases slightly acid or even alkaline gastric contents were frequently observed. Furthermore, the real grave symptoms of increased secretion of juice, the severe burning, the sensation of caustic acid, and the self-evident relief after evacuation of the abnormal fluid, are missing. I agree with Bouveret, who considers it as doubtful whether a distinction of a special form of flow of gastric juice of central origin is justified.

Often a hysteric or neurasthenic form of flow of gastric juice has been spoken of. Indeed, according to the experiences of others and my own, we have to deal with more or less nervous individuals, but it remains still undecided whether the flow of gastric juice is to be considered as a partial symptom of the neurasthenic condition, or rather as coincident.

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[150] Rossbach, Deutsches Archiv für klinische Medicin, 1885, Bd. 35.

[151] v. Noorden, Charité annalen, 1890.

[152] Boas, Deutsche medicinische Wochenschrift, 1889, No. 42.

[153] Bouveret, Les maladies de l'estomac, Paris, 1893, P. 680.

[154] Seymour Basch, Archiv für Verdauungskrankheiten, 1899, Bd. 5, P. 29.

Gastroxynsis, so-called by Rossbach, is stated to
persons of the well-educated class, especially in les
ing causes are said to be emotional irritations, wr
tion (abuse of tobacco). Rossbach explains the sy
fact that under certain conditions on the one sid
secretion of gastric acid is incited through partly
impulses upon the vagus current; and on the c
stronger reflexes are liberated in more distinct r
acting upon the sensory gastric nerves, than in c
system. M. Rosenthal,[155] however, refers the sym
lations of the central vasomotors, which cause, besid
anæmia, an irritation of the bulbar emetic center
tric secretion.

Diagnosis.

The diagnosis is based upon the fact that the
in paroxysms, sometimes during the best of health
described character of the attacks, and finally, up
the aspirated or vomited gastric contents.

The attacks may be preceded by premonitory s
ache, nausea, lassitude, feeling of heaviness all ove
the attacks start early in the morning with anorexi
perhaps severe headache. These symptoms are f
of intolerable burning, cramp-like and most exha
patients themselves usually attribute to the supe
ducts. The paroxysm may occasionally be checked
of alkalies or by drinking large quantities of wat
done, at the height of the attack there occurs vom
clear as water, strongly acid, setting the teeth on ed
admixture of bile and mucus; after that there is pe
cases. In other cases the paroxysm is followed by n
tioned, may continue for several days or even tw
time the patients feel as prostrated as those sufferi
There exist severely burning gastralgia, absolute a
ness, constipation, oliguria. The patient looks ex
abdomen is sunk in, the pulse is hard and usuall
ever, patients suffering from gastric crises are refr
and enabled to endure the recurrence of the attac

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[155] M. Rosenthal, Magenkatarrh und Magenneurosen, Wi

the day, patients with flow of gastric juice are unceasingly disturbed through unbearable burning and nausea, and they are compelled to find relief by morphine.

The *objective* finding, aside from the examination of the vomited material, does not yield much: a sensation of slight pain in the epigastrium during the paroxysms is all that can be found, except the symptoms and sequelæ of the paroxysm. The vomit at the beginning of the attack is clear, thin, odourless, mixed with bile or mucus, its reaction very acid. In one case Rossbach could detect an amount of hydrochloric acid of 4 per mille. Later the vomit may have a large admixture of bile, whereby part of the original acidity becomes neutralised. Occasionally traces of blood are also found in the vomit, which may suggest the thought of gastric ulcer to the inexperienced.

In several cases of mine (see below) I could demonstrate that the secretion of acid is increased and that the flow of gastric juice may be observed to a greater or lesser extent, though not constantly, also in the intervals. Moreover, I could not convince myself in a case observed for a long time (Case II) that the quantities of gastric juice were considerably larger during the paroxysms than in the intervals. Is it not possible that the continuous absorption of the strongly acid and increased gastric juice into the blood could be found the inciting factor of the paroxysm?

In the following I report a few marked clinical histories, referring on the one side to the periodic flow of gastric juice, and on the other side to Rossbach's gastroxynsis:—

CASE I.—L. H., merchant, 46 years of age, of healthy family, and always enjoyed good health except his present suffering, which has lasted for twenty years. He refers the trouble to the irregular mode of life to which he was subjected while a travelling salesman. He admits particularly to have eaten and drank hastily. Other errors of diet are also admitted, although patient is not addicted to drinking or smoking. But in the last few years he has become "somewhat nervous." The patient's disease occurred periodically, about every three or four months. In the intervals his condition was satisfactory, especially when he was careful in eating and drinking. This intelligent patient describes the paroxysms as follows: The first symptoms are eructations, loss of appetite, nausea and pressure, and finally vomiting. The vomit rarely contains food, and usually consists of large quantities (one litre and more) of a watery, intensely acid fluid, so that the teeth are set on edge. The vomiting lasts for three or four days; the whole attack one to two weeks. After the vomiting, severe pains in the epigastrium develop, which radiate towards the back, and occasionally also towards the shoulders. The urine was dark and scanty. Strength became very much reduced; patient was compelled to remain in bed; the gastralgias did not cease during night-time, so that patient repeatedly required injection of morphine. The pains gradually ceased; patient recuperated and could again attend to his business. Such was the course of the trouble for seventeen years. In the last three years patient used the waters of Carlsbad with good effect. In 18 patient again went to Carlsbad, but returned in a decidedly worse condition; Carlsbad severe gastralgias and anorexia made their appearance; in connection wi them were sleeplessness and irregular movements of the bowels. On his return fr Carlsbad the above-described vomiting occurred. In this condition patient ca under my observation. *Status præsens:* very much emaciated; normal respirato and circulatory organs. Stomach, when empty, in collapsed condition, no spla sound. In the epigastrium, diffuse painfulness (algesimeter, 8 kilos); no dor tenderness. Stomach-contents examined twice after fasting, and twice after te breakfast. From the fasting stomach, the first time 25 cubic centimetres, the seco time 65 cubic centimetres of a slightly cloudy juice (with respective acidity of and 85) were recovered.

Acidity after test-breakfast was 90 and 86. No particular signs of motor sufficiency. Essential improvement through continued use of magnesia ammon phosphorica, 2.0 three times a day.

CASE II.—Miss R., 24 years of age. Mother died of cancer of the stomac father healthy. Patient, when still in childhood, suffered from occasional vomitin appetite was good in the intervals. Menses, when 14½ years old, every four or fi weeks, painful; bowels slightly constipated. For ten years the trouble has develop as follows: when the stomach is empty, burning in the epigastrium occurs frequent radiating towards the throat; pain is relieved by ingestion of food; but after eati there appears great thirst, so that patient has to drink very much water, where a sensation of fulness is caused. Occasionally patient has a "rolling" sensation the abdomen. Appetite good, patient can eat any kind of food with impunity, p haps greasy food excepted. Every week, sometimes every other or every third we there occurs, with definite prodromal symptoms, a paroxysm, lasting about for eight hours. The prodromal symptoms are cramps and pain in the epigastrium, a excessive, voracious hunger, so that patient must continually take food. T paroxysm itself starts with severe headache and gastralgia, nausea, and dizzine This condition is relieved by belching of air. With absolute rest of the patient t paroxysm may remain at this stage. But when patient moves, vomiting very so occurs; the vomit is usually of green colour and tastes very sour. The vomiti is repeated every half hour in severe paroxysms, otherwise every two or three hou On the day of the paroxysm patient cannot eat anything. There is severe headac sometimes hemicrania, other times over the whole head. Only at night-time, wh patient falls asleep, improvement. The following day patient can again attend her duties. Patient has lost eight pounds in last few weeks. *Status præsens:* pall moderately well-nourished girl. At the apex of the heart slight murmur, audi especially after quick bending forward. Extremities cool. Stomach slightly ten on pressure, but not painful. No splash sound in fasting stomach; only after dri ing of 150 cubic centimetres of water splashing about at the level of the umbilic Examination of stomach-contents:—

(*a*) In the intervals free from attacks, repeatedly 80 to 100 cubic centimet of a clear, water-like fluid of intensely acid reaction are recovered from the fasti stomach. Strong HCl reaction, 2.4 to 2.6 per mille.

(*b*) During paroxysm, repeatedly 100 to 250 cubic centimetres of bile-colour intensely sour masses are expressed from the fasting stomach; acidity, 2.5 to 2.8 mille HCl.

(*c*) After test-breakfast, acidity 2.6 per mille HCl.

CASE III.—Anton B., 35 years of age, machinist. He suffered, in 1887 and 1888, from attacks of dizziness and dyspnœa, which patient attributed to an abnormal gain in weight. Patient used Carlsbad salt for this trouble. Since that time digestive disorders. Patient could not ingest meat-broth or beverages, as they were always vomited. A short time later there occurred "stomach acidity," associated with headache and vomiting of a sour, acrid, slimy fluid. Great reduction of weight followed; sleep was interrupted. In the intervals also the stomach is not normal, because a stomach acidity appears after ingestion of milk and cocoa.

Patient describes the paroxysms as follows: In the morning on arising occasional eructations; towards noon, slight pressure in neck and back of head, becoming exacerbated after dinner, affecting the whole head toward evening, and unbearable, especially when lying down. The paroxysm is checked only after vomiting or eructation (expression) of large quantities of acidity, or through washing out the stomach. After the acid taste has been decreased by administration of large doses of sodium bicarbonate, a clear, slimy, and ropy fluid is expressed. Patient denies special mental strain or overwork, or abnormal excitement, but admits general nervousness. The physical examination of the stomach in the intervals yielded entirely normal conditions; repeated analysis of the gastric contents after test-breakfast showed HCl values between 2.29 and 2.68 per mille.

Differential Diagnosis.

In this respect we have to consider:—

(*a*) Gastric crises (see page 670).

(*b*) Periodic vomiting (see page 671).

Treatment.

If mental strain is present, prophylactic measures are of the utmost importance: long and absolute avoidance of mental work, with sojourn in the country or in the mountains; increased bodily action (turning, swimming, fencing, riding on horse-back, gymnastics, [golf]. When emotional excitement or abuse of tobacco can be considered as exciting causes, they must be avoided.

As to the paroxysm itself, early mechanical removal of the acidity by means of expression or lavage (alkalies) is occasionally effective. When, as in the above-described cases, the affection originates in a chronic superacidity or gastrochylorrhœa, the measures (see page 537), usually of value in such conditions are to be employed.

(4) *Nervous Dyspepsia.*[156]

(Neurasthenia Gastrica.)

Preliminary Remarks.—By nervous dyspepsia the authors who have paid special attention to this subject (v. Leube, Stiller, Ewald, Oser, v. Leyden, and others) understand a great variety of conditions. v. Leube limited the clinical picture to the narrowest limits in describing as nervous dyspepsia a picture in which anatomical changes were absent. In more recent time, however, v. Leube goes farther in describing it first as a symptom-complex in which the nerves supplying the digestive organs are affected by anatomical changes of the stomach and the consecutive altered chemism of digestion, and second as a symptom-complex of nervous dyspepsia, in which, in spite of a normal anatomic condition of the stomach, an abnormal state of irritation is caused by the corresponding irritated nerves. There is, however, undoubtedly a third reflectory form of nervous dyspepsia which originates in other organs, such as the kidneys, uterus, ovaries, the male generative organs, and particularly in the intestines. But constitutional diseases also, namely, anæmia, diabetes mellitus, tuberculosis, [syphilis], etc., may furnish the foundation for the development of nervous dyspepsia. Nervous dyspepsia, therefore, is not a disease, but a symptom-complex, in which organic changes connected directly or indirectly with the digestive organs may be detected or may be absent. It might be useful to speak of an idiopathic and a deuteropathic nervous dyspepsia, whereby, of course, we must remember that the idiopathic form is justified only as long as our knowledge about the origin of the gastric neuroses has no surer foundation.

There were also discussions whether the nervous dyspepsia is an independent neurosis or a partial symptom of general neurasthenia or of hysteria. According to my experience, the two forms may occur, and I agree with those writers who admit an independent primary gastric disturbance which is not complicated, or apparently not complicated, with general neurasthenia (Rosenheim, L. Herzog,[157] and others).

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[156] *Literature.*—v. Leube, Deutsches Archiv für klinische Medicin, 1879, Bd. 23; Verhandlungen des III Kongresses für innere Medicin, 1884 (References); Spezielle Diagnose innerer Krankheiten, Bd. 1.
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Glax, Volkmann's Sammlung klinische Vorträge, 1882, No. 223.
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E. v. Leyden, Berliner klinische Wochenschrift, 1885, No. 30.
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Saupault, Les dyspepsies nerveuses, Paris, 1893.
Rosenheim, Berliner klinische Wochenschrift, 1897, No. 42-44.
Honigmann, Zeitschrift für praktische Aerzte, 1897, No. 24.
Verhaegen, Sur les troubles digestifs des hystériques, Brussels, 1896.
Compare also text-book on Diseases of the Stomach.

In nervous dyspepsia not only one, but several, functions of the stomach are affected, namely, the sensory as well as the motor and secretory functions may become disordered. Furthermore, it must be remembered that organic and functional disturbances may be synchronous, that, for instance, symptoms of nervous dyspepsia may be associated with chronic gastritis, ulcer, atony, etc. It will be sometimes very difficult to decide which is the primary and which is the secondary affection, as under certain circumstances organic affections, such as atony or gastroptosis, may follow nervous dyspepsia. Still, it must not be overlooked that the conception of nervous dyspepsia, as Ewald has emphasised, concerns not only the stomach, but affects the entire alimentary tract, and may even affect the heart and circulatory system (palpitation of the heart, pseudo-angina, epigastric pulsation, nervous asthma, etc.).

Diagnosis.

The diagnosis of nervous dyspepsia is very easy in some cases, difficult but still entirely attainable in other cases, and in still other cases quite impossible.

As to diagnosis, we must consider as important, first, the detection of general neurasthenia, then the troubles affecting the digestive tract in particular. The objective examination may be entirely negative, or we may find an organic affection situated in the digestive sphere or being in some connection with the same. We must turn particular attention to displaced kidneys, enteroptosis, entozoa, small tumours, hernia (epigastric hernia), pathologic changes of the male or female genito-urinary apparatus, organic affections of stomach and intestines, etc. Usually there exists an evident disproportion between the possible objective changes and the complaints of the patients. Furthermore, the independence from or the loose connection of the dyspeptic complaints with quality and quantity of the ingesta is characteristic, while on the other hand, their connection with other factors, quite extraneous to the digestive function (sleep, work, environment, climate, etc.), is pronounced.

As regards the subjective complaints, they depend on the abnormal reaction of the digestive apparatus upon purely physiologic stimuli, as w as on the influences provoked by this reaction, which, on their part aga affect unfavourably the nervous system.

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[157] L. Herzog, Zeitschrift für diätetische und physikalische Therapie, 1898, Bd. 2, P. 116.

When the stomach is empty the patients complain of pressure in t head, lassitude, weakness, loss of ambition, palpitation of the heart, as th cannot eat or do not want to eat; and when the stomach is full, the ing tion of food provokes dyspeptic complaints and the above-mentioned ir tative symptoms of the sensory sphere.

The variety of the gastric troubles is exceedingly protean, and t colouring which the patients give in the description of the trouble deper not a little on the degree of their education and their imagination. Besi the usual dyspeptic disorders, we are told of a feeling of "hot tinglin of "abnormal flushes," of stinging, pricking, gnawing, burning, bori beating sensations. In a case of severe gastric neurasthenia under observation, the patient had the feeling as if "five hundred thousand devi were playing in her abdomen. Another had the sensation as if the int tines were put into a vise, etc. In this I might say exaggerated descr tion, which involuntarily recalls the loud colouring of the modern realis art in painting, I find an important criterion of a functional disturban which exists, of course, but which is felt as excessive through the morl irritative condition. In remarkable contrast is the fact that the patie occasionally, without any evident explanatory causes, feel better, sh better appetite and sleep, and gain in weight, while on the other ha just such periods of deterioration immediately follow. In some patie the conditions vary from week to week, yes, from day to day. In c nection with this is the observation that also objective controllable gast and intestinal functions (nervous constipation, nervous diarrhœa) are fluenced in the one or the other direction through emotional excitem or depression. In this very condition of nervous dyspepsia, for instan the most varied degrees of hydrochloric acid secretion are detected; likew the motor function may vary in certain limits. In this respect I ment the following examples:—

CASE I.—Francis P., clerk, 22 years old, suffering from gastric troubles si the summer of 1889. The complaints refer to pressure and fulness after eati nausea, eructation of air. Bowels slightly constipated. States he has lost sc weight, but he looks well. Stomach slightly tender on pressure; greater curvat at the level of the umbilicus.

The chemical examination shows: The first day no free HCl, no Uffelmar reaction. The following day free HCl distinct, rennin-zymogen positive $^1/_{100}$. Th weeks later abundant free HCl (Congo titration, 0.18 per cent.). A year later free HCl. After regulation of diet and administration of HCl for a few weeks, HCl returned (Congo 0.08 per cent.); two weeks later no free HCl. (The exami tions were always made precisely an hour after test-breakfast.) Repeated tests for ferments showed a constant amount of lab-zymogen of $^{1}/_{100}$.

CASE II.—Mrs. H., 30 years of age, healthy family; formerly always well; menses since her fourteenth year; two normal confinements; according to her statement peritonitis after the first child-birth. Complains of feeling of oppression in the gastric region, which has existed for many years. It increases immediately after eating. Appetite good, never vomiting, no sensation of pain, urine clear, bowels regular. Occasional headache, especially after excitement. Great thirst. Patient often has fits of crying without special cause. Epigastrium quite tender on pressure. Stomach of normal position and size. Heart and lungs normal, no albumen or sugar in urine. The examination of the gastric contents on the first day does not show free HCl, on the second day, a small amount (0.05 per cent.); two weeks later, 0.116 per cent. (Congo) HCl with marked colour reaction; a week later, 0.13 per cent.; after a period of another two weeks *no free HCl*, but lab-zymogen up to $^{1}/_{100}$.

CASE III.—Elizabeth B., 17 years of age, servant girl; formerly well. Since January, 1900, dysmenorrhœa, cystitis. For eight weeks gastric disturbances, such as eructation of air, fulness, pressure, and pain in the epigastrium; food is often regurgitated and ejected. Sometimes nausea and vomiting; bowels very much constipated, move only after the use of purgatives. *Status præsens:* cystitis, abdomen distended, contents of colon markedly palpable, otherwise normal conditions. Four individual examinations after test-breakfast yield as follows: August 1, 1901, an hour after test-meal, small amount (8 cubic centimetres) of contents; Günzburg and resorcin tests give weak, positive reaction. August 2, expression does not produce any contents. August 3, 25 cubic centimetres filtrate, HCl very distinct (Congo acidity = 0.146 per cent.). August 4, abundant stomach-contents, free HCl distinct (Congo acidity, 0.18 per cent.). Without any treatment whatsoever appetite improved, eructation ceased.

These variations in the functional activities are an exceedingly important objective sign for the existence of a nervous dyspepsia It is true, they are met with also in organic gastric diseases, but never in such a rapidly changing manner.

v. Leube has taught us another important objective sympton, namely, the emptiness of the stomach seven hours after the ingestion of the test-meal suggested by him. There are, of course, not infrequent exceptions to this average period, for conditions of slight atony of the stomach are not at all rare; but as a whole the said method fulfils its purpose of demonstrating that a grave motor obstacle is not present.

The most important and, according to my opinion, most decisive symptom is the completely intact condition of the motor and secretory function of the stomach in spite of the most pronounced sensory disturbances (pressure, fulness, distension, eructation, anorexia, etc.). If such conditions are synchronous we will hardly fail in the diagnosis. *Vice versa,* our judgment becomes more difficult in the presence of motor and secretory disorders. Is there an anatomic affection present, or does a nervous trouble exist, or have we to deal with a combination of the two conditions? The decision is uncommonly difficult in spite of the greatest painstaking efforts and experience, and generally valid rules can hardly be given for the numerous combinations in question.

According to my opinion, only careful observations in reference to subjective complaints as well as objective changes can protect us from mistakes, and above all I recommend paying most particular attention to the variations of the intensity and qualities of the complaints, as well as to the said protean changes of secretion and motility. Nevertheless, there will be cases which either cannot be explained at all, or perhaps only after a long period, or finally find an explanation in a most unfortunate manner through an autopsy. These are the cases in which we are surprised by a slightly marked Addison's disease or by carcinomatous or tuberculous condition of the peritoneum, by a carcinoma of the pancreas, or also by a latent cancer of the stomach. Nobody, not even the most skilled diagnostician, will fail to experience unpleasant running off the tracks of this kind. Such conditions ought to teach us to be more cautious and guarded in making the diagnosis of nervous dyspepsia than is still frequently the case.

Differential Diagnosis.

In this respect we have particularly to consider chronic gastritis, atony of the stomach, and occasionally also ulcer and carcinoma.

The most important differential signs between nervous dyspepsia and chronic gastritis have already (page 416) been discussed. As to differential diagnosis between nervous dyspepsia and myasthenia, see page 483.

The differential diagnosis between nervous dyspepsia and ulcer can evidently be a question only in cases of ulcer without preceding hæmorrhage. In these cases, however, the constant gastric pain usually dependent upon the ingestion of food, as well as the circumscribed epigastric and dorsal pain, are useful, though not irrefutable, differential signs (page 443). In contrast to them are the irregularly arranged pressure and pain points in nervous dyspepsia. On the other hand, it has been emphasised that as result of an ulcer and an existing predisposition, disturbances may develop which greatly simulate nervous dyspepsia, examples of which I have described above (page 435).

Concerning differential diagnosis between nervous dyspepsia and gastric carcinoma, see page 592.

Treatment.

First of all the connection of nervous dyspepsia with other morbid conditions is to be considered, whereby the above-mentioned (page 702) momenta have to be carefully watched. Thus, for instance, if eggs or parts of a tape-worm are found, a cure for tape-worm* will be the suitable treatment; in nephroptosis, a well-fitting bandage and rest-cure, [nephropexy]; in anæmia, a regular milk and iron treatment; in gastritis, or atony, or enteritis, a corresponding therapy will come under consideration. We must not neglect, besides the real genital affections, the perversities of the sexual life such as exhibited in a most terrible degree in modern times.

If no special cause can be found and we must deal, therefore, with the idiopathic form of nervous dyspepsia, the general neurasthenia must be the first subject to be attacked. In such cases those principles are to be considered which are important also otherwise in the treatment of the constitutional neuroses, namely, dietetics, mechanotherapeutics, electrotherapeutics, hydropathic and balneotherapeutic curative methods.

The suitable measure in the individual case is to be conformed to the variety of the affection. We often learn from the failures of the patients what we should not employ, and, however paradoxical it may sound, that physician has the highest trump in his hand in the treatment of the neurasthenic in general and of the nervous dyspeptic in particular, who treats them as the last.

It is an absolutely impossible task to enumerate all those curative measures which may be suitable to free nervous dyspeptics from their suffering. I can give only a sketch of those methods which, in my experience, usually prove themselves of value. First, we must improve the nutrition of the patient, which usually had been considerably affected; then we must consider the special diet suitable to the condition of the gastroenteric canal. Every trained physician has formed his own opinions in this regard, and we may easily observe that many methods of various kinds lead to the desired effect.

At the present time there is general agreement at least in that point that strict dietetic measures are nowhere less out of place than in the neurasthenic dyspeptic. All principles of dietetics are shattered at the protean and varied conditions of the digestive disturbances. *In these cases a systematic experimenting has the value of a scientific method.* When milk and cream are liked and well borne, it is undoubtedly one of our most efficient allies in the battle against the stubborn complaint. But frequently enough we must renounce it. We will, however, not neglect to emphasise that in many cases a gradual decrease of intolerance to milk may be observed. The varied preparation of milk (kefyr, kumyss, sour milk, buttermilk, milk or cream soups, rice, farina, and other cereals in milk, offer so much change that the milk cure is felt to be less monotonous.

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* A case of cure of a nervous dyspepsia through removal of a tape-worm was recorded by Senator (Kongress für innere Medicin, 1884). I have met several cases of similar kind in my own practice).

In the selection of other foods the patient should be allowed to have as mu choice as possible; his inclinations and aversions ought to be studied, a we shall gain his confidence—a most important point—much more th by prescribing for him a new medicine every day.

In every case monotony of the kitchen must be severely avoided. T food must be sufficiently seasoned. Roast meat is borne better than boil meat. The relishes, too, which often are neglected, are a most necessa even indispensable aid of the dietetic therapy in the neurasthenic. Go fruit, grapes, dates, figs, raisins, etc., are the more indicated as they sim taneously fill an important indication, namely, the regulation of the bowe As regards the latter, we advance the same principles as in habitual co stipation, namely, that nothing is less advisable than purgatives. Besid the corresponding diet, abdominal massage, free exercises (physical c ture), energetic faradisation of the abdominal walls, or the still more fective faradisation by means of the rectal electrode, are to be recommende In stubborn cases injection of olive oil, codliver oil and soda, castor oil, et are usually quite satisfactory.

Alcohol in form of good beer and wine (the heavy Spanish and Gre wines excepted) may be allowed in moderate amount, but as a whole ought to be very cautious in the administration of alcoholica in the neura thenic.

In especially stubborn cases of nervous dyspepsia a *Weir Mitchell cu* is frequently of great and permanent benefit, but as not a few cases g worse under this rest- and feed-cure, we must here recognise the necessi of strict individualisation. According to my experience, the most suitab cases for rest- and feed-cure are those in which the state of nutrition h been considerably reduced and insomnia is present, while the digesti organs appear to be objectively normal. But in irritable and restle patients, or in cases of disturbances in the course of digestion, only mo erate effects or none at all will be gained. That, however, in rest-cur the individual influence of the physician plays a decisive part and ove comes great difficulties, even in desperate cases, is a fact well known experienced physicians.

Mechanotherapy consists essentially of massage, partly local, as abdon inal massage, and partly general, as body massage. As to the effect abdominal massage, compare Part I, page 330. To the mechanical trea ment belongs also the application of the tube. In nervous dyspepsia, as almost all other gastric affections, lavage has been tried, and occasional good results have been obtained. As a whole, however, we cannot expe very much from lavage, unless we employ it as a suggestive agent. Mo suitable and effective in some cases is the gastric douche, as recommende first by Malbranc (Part I, page 353). It is especially Rosenheim,[158] who employs the gastric douche in nervous dyspepsia with or without general neurasthenia.

As in nervous gastric affections in general, hydrotherapeutics in various forms and applications play also an important aiding part. The treatment in a sanatorium is the most useful one, because here the daily observation facilitates the examination of the reaction of the patient under thermic influences and their necessary gradation. In milder cases the domestic use of hydrotherapeutics in form of ablutions, rubbing, packs, douches, etc., may be accompanied by success.

Likewise electric and gymnastic methods, separately or in combination with the above-mentioned measures, are to be employed.

As to *medicines*, there are especially four groups to be considered: stomachics, tonics, sedatives, and hypnotics. The first-mentioned have no especially excellent effects; perhaps the best are the extract of nux vomica (0.01 to 0.05, in powders or pills), condurango and columbo, and bela Indica in form of fluid extracts (teaspoonful three times a day), orexine tannate (0.25 pro dosi). Elixir of orange, compound tincture of quinine, and similar compounds are of less value. In severe cases of anorexia almost all medicines are unavailing.

In nervous dyspepsia due to anæmia the numerous iron preparations, quinine, and arsenic (arsenious acid, 0.001 to 0.0015 in granules) are worth a trial. A suitable form of administration of arsenic and iron is the waters of Levico and Roncegno (1 tablespoonful in a wineglassful of water, two or three times a day).

The bromides are first among the sedatives, especially given in infusion of catnip or tea of orange-blossoms. They may be prescribed as follows:—

℞ Ammonii bromati,
Natrii bromati .... aa 1.0
M. ft. pulv.
Dentur tales doses No. xx.
Sig.: One powder in a cup of tea of catnip or orange blossoms in the morning and evening.

[Strontium bromatum may be highly recommended.]

Validol (a combination of valerian and menthol), first recommended by Schwersenski,[159] has proved a good palliative remedy in nervous dyspepsia. The dose is 8 to 10 drops in sweetened water three times a day.

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[158] Rosenheim, Therapeutische Monatshefte, August, 1892, P. 382.

[159] G. Schwerenski, Therapeutische Monatshefte, November, 1897.

The combination of ferrum bromatum and chininum bihydrobr micum, as recommended by Maximowitch in neurasthenia due to anæmi has been frequently of good service:—

℞ Ferri bromati,
Chinini bihydrobromic. .... aa 4.0
Extracti et pulveris radic. rhei .... q. s.
M. fiant pilul. No. cx.
Sig.: Two pills three times a day.

Opiates are best to be avoided, on account of the unfavourable influen upon the alimentary canal; perhaps the codeine preparations, whi inhibit but slightly the intestinal peristalsis, may be employed.

Ewald[160] some years ago recommended chloral hydrate and caffei chloral as a good sedative; more recently Rosenbach[161] recommends it doses of 0.1 to 0.2. The latter author particularly emphasises the favou able influence of chloral upon the removal of the disagreeable sensatio of anxiety and pressure as well as of flatulency.

Hypnotics cannot be avoided in cases of pronounced agrypnia. T most suitable is chloral hydrate, administered by the rectum in doses of 3 or 4.0 grammes in an appropriate vehicle. [While the doses of 3.0 or 4 have often been used without the least trouble, we ought to be very car ful, since even smaller doses may cause dangerous symptoms.] Internall chloralamid (in powders of 1.0 to 2.0 grammes), sulphonal (1 to grammes), and trional (1 to 2 grammes) have shown themselves as usef hypnotics. [Veronal and somnos have been of good service to me various cases.]

Mineral waters may under some circumstances be of help, especial the muriatic iron waters (Franzensbad, Pyrmont, Elster, Cudowa, Sclera bach. Rippoldsau, Petersthal, Griesbach, Freyersbach, etc. [Chalybea Spring, Bedford, Pa.]), are of use, but treatment of this kind is indicate only in the milder cases of nervous dyspepsia; in grave cases they are wit out effect. The same is to be said of climatic changes in the mountai and at the sea-shore, unless they are combined with treatment in a san torium with its modern apparatus. As in other gastric neuroses, the su phate of soda waters of Carlsbad, Marienbad, etc., have rather an unfavou able influence upon nervous dyspepsia; while, on the other hand, I occ sionally saw a favourable effect from chloride of sodium waters, such Kissingen, Homburg, Wiesbaden.

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[160] Ewald, Klinik der Verdauungskrankheiten II, 3 Aufl., S. 548 and 552.

[161] O. Rosenbach, Therapeutische Monatshefte, September, 1899.

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